Ref #	Hits	Search Query	DBs	Default Operator	Plurals	Time Stamp
L1	87	(lectin or hemaggluinat$ or ricin) near3 resist$8	US-PGPUB; USPAT	OR	OFF	2004/06/01 11:28

PGPUB-DOCUMENT-NUMBER: 20040093621

PGPUB-FILING-TYPE: new

DOCUMENT-IDENTIFIER: US 20040093621 A1

TITLE: Antibody composition which specifically binds to CD20

PUBLICATION-DATE: May 13, 2004

INVENTOR-INFORMATION:

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APPL-NO: 10/ 327663

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COUNTRY	APPL-NO	DOC-ID	APPL-DATE
JP	2001-392753	2001JP-2001-392753	December 25, 2001
JP	2002-106948	2002JP-2002-106948	April 9, 2002
JP	2002-319975	2002JP-2002-319975	November 1, 2002

US-CL-CURRENT: 800/6, 435/334

ABSTRACT:

The present invention provides an antibody composition which specifically binds to CD20 and comprises an antibody molecule which has complex N-glycoside-linked sugar chains bound to the Fc region; a process for producing the antibody composition; and a medicament comprising the antibody composition.

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Summary of Invention Paragraph - BSTX (64):
[0062] (e) a technique for selecting a cell line **resistant to a lectin** which recognizes a sugar chain in which 1-position of fucose is bound to 6-position of N-acetylglucosamine in the reducing end through .alpha.-bond in the complex N-glycoside-linked sugar chain.

Summary of Invention Paragraph - BSTX (65):
[0063] (15) The cell according to any one of (1) to (14), which is **resistant to at least a lectin** which recognizes a sugar chain in which 1-position of fucose is bound to 6-position of N-acetylglucosamine in the reducing end through .alpha.-bond in the complex N-glycoside-linked sugar chain.

Brief Description of Drawings Paragraph - DRTX (18):
[0148] FIG. 17 shows a result of measurement of the binding activity of an anti-CD20 chimeric antibody R92-3-1 produced by **lectin-resistant** CHO/DG44 cell while changing the concentration of the antibody using the immunofluorescent

method. The ordinate and the abscissa show the relative fluorescence intensity at each concentration and the antibody concentration, respectively. ".box-solid." and ".smallcircle." show the activities of Rituxan.TM. and R92-3-1, respectively.

Brief Description of Drawings Paragraph - DRTX (19):

[0149] FIG. 18 shows a result of the evaluation of ADCC activity of the anti-CD20 chimeric antibody R92-3-1 produced by lectin-resistant CHO/DG44 cell, using Raji cell as the target cell. The ordinate and the abscissa show the cytotoxic activity on the target cell and the antibody concentration, respectively. ".box-solid." and ".smallcircle." show the activities of Rituxan.TM. and R92-3-1, respectively.

Brief Description of Drawings Paragraph - DRTX (20):

[0150] FIG. 19 shows an elution pattern obtained by reverse phase HPLC analysis of a PA-modified sugar chain prepared from the anti-CD20 chimeric antibody R92-3-1 produced by lectin-resistant CHO/DG44 cell. The ordinate and the abscissa show the relative fluorescence intensity and the elution time, respectively. Analytical conditions of the reverse phase HPLC, identification of the sugar chain structure and calculation of the ratio of sugar chains to which .alpha.1,6-fucose was not bound were carried out in the same manner as in Example 3.

Detail Description Paragraph - DETX (69):

[0226] (e) a technique for selecting a cell line resistant to a lectin which recognizes a sugar chain in which 1-position of fucose is bound to 6-position of N-acetylglucosamine in the reducing end through .alpha.-bond in the complex N-glycoside-linked sugar chain.

Detail Description Paragraph - DETX (221):

[0378] (5) Method for Selecting a Cell Line Resistant to a Lectin which Recognizes a Sugar Chain Structure in which 1-position of Fucose is Bound to 6-position of N-acetylglucosamine in the Reducing End Through .alpha.-bond in the N-glycoside-linked Sugar Chain

Detail Description Paragraph - DETX (222):

[0379] The host cell for preparing the cell of the present invention can be prepared by using a method for selecting a cell line resistant to a lectin which recognizes a sugar chain structure in which 1-position of fucose is bound to 6-position of N-acerylglucosamine in the reducing end through o-bond in the N-glycoside-linked sugar chain.

Detail Description Paragraph - DETX (223):

[0380] The method for selecting a cell line resistant to a lectin which recognizes a sugar chain structure in which 1-position of fucose is bound to 6-position of N-acetylglucosamine in the reducing end through .alpha.-bond in the N-glycoside-linked sugar chain includes the methods using lectin described in Somatic Cell Mol. Genet., 12, 51 (1986) and the like.

Detail Description Paragraph - DETX (225):

[0382] Specifically, the cell line of the present invention resistant to a lectin which recognizes a sugar chain structure in which 1-position of fucose is bound to 6-position of N-acetylglucosamine in the reducing end through .alpha.-bond in the N-glycoside-linked sugar chain can be selected by culturing cells for 1 day to 2 weeks, preferably from 1 day to 1 week, using a medium comprising the lectin at a concentration of 1 .mu.g/ml to 1 mg/ml, subculturing surviving cells or picking up a colony and transferring it into a culture

vessel, and subsequently continuing the culturing using the lectin-containing medium.

Detail Description Paragraph - DETX (486):
[0637] Preparation of Lectin-Resistant CHO/DG44 Cell and Production of Antibody Using the Cell:

Detail Description Paragraph - DETX (487):
[0638] 1. Preparation of Lectin-Resistant CHO/DG44

Detail Description Paragraph - DETX (488):
[0639] CHO/DG44 cells were grown until they reached a stage of just before confluent, by culturing in a 75 cm.sup.2 flask for adhesion culture (manufactured by Greiner) using IMDM-FBS(10) medium MOM medium comprising 10% of fetal bovine serum (FBS) and 1.times. concentration of HT supplement (manufactured by GIBCO BPL)]. After washing the cells with 5 ml of Dulbecco's PBS (manufactured by Invitrogen), 1.5 ml of 0.05% trypsin (manufactured by Invitrogen) diluted with Dulbecco's PBS was added thereto and allowed to stand at 37.degree. C. for 5 minutes to dissociate the cells from the flask bottom. The disociated cells were recovered by a centrifugation operation generally used in cell culture and suspended in IMDM-EBS(10) medium at a density of 1.times.10.sup.5 cells/ml. To the cell suspension, and then 0.1 .mu.g/ml of an alkylating agent N-methyl-N'-nitro-N-nitrosoguanidine (hereinafter referred to as "MNNG", manufactured by Sigma) may be added, if necessary. After incubating them at 37.degree. C. for 3 days in a CO.sub.2 incubator (manufactured by TABAI), the culture supernatant was discarded, and the cells were again washed, dissociated and recovered by the same operations, suspended in IMDM-FBS(10) medium and then inoculated into a tissue culture 96 well plate (manufactured by IWAKI Glass) at a density of 1,000 cells/well. To each well, as the final concentration in medium, 1 mg/ml Lens culinaris agglutinin (hereinafter referred to as "LCA", manufactured by Vector), 1 mg/ml Aleuria aurantia agglutinin (Aleuria aurantia lectin; hereinafter referred to as "AAL", manufactured by Vector) or 1 mg/ml kidney bean agglutinin (Phaseolus vulgaris leucoagglutinin; hereinafter referred to as "L-PHA", manufactured by Vector) was added. After culturing them at 37.degree. C. for 2 weeks in a CO.sub.2 incubator, the appeared colonies were obtained as lectin-resistant CHO/DG44. Regarding the obtained lectin-resistant CHO/DG44, an LCA-resistant cell line, an AAL-resistant cell line and an L-PHA-resistant cell line were named CHO-LCA, CHO-AAL and CHO--PHA, respectively. When the resistance of these cell lines to various kinds of lectin was examined, it was found that the CHO-LCA was also resistant to AAL, and the CHO-AAL was also resistant LCA. In addition, the CHO-LCA and CHO-AAL also showed a resistance to a lectin which recognizes a sugar chain structure identical to the sugar chain structure recognized by LCA and AAL, namely a lectin which recognizes a sugar chain structure in which 1-position of fucose is bound to 6-position of N-acetylglucosamine residue in the reducing end through .alpha.-bond in the N-glycoside-linked sugar chain. Specifically, it was found that the CHO-LCA and CHO-AAL can show resistance and survive even in a medium supplemented with a pea agglutinin (Pisum sativum agglutinin; hereinafter referred to as "PSA", manufactured by Vector) at a final concentration of 1 mg/ml. In addition, even when the alkylating agent MNNG was not added, it was able to obtain lectin-resistant cell lines by increasing the number of cells to be treated. Hereinafter, these cell lines were used in analyses.

Detail Description Paragraph - DETX (490):
[0641] Into 1.6.times.10.sup.6 cells of the CHO/DG44 cell which was the lectin-resistant cell line obtained in the above item 1, 4 .mu.g of an anti-CD20 vector for expression of human chimeric antibody pKANTEX2B8P was introduced by electroporation [Cytotechnology, 3, 133 (1990)], the cells were

suspended in 10 ml of IMDM-dFBS(10)-HT(1) [IMDM medium (manufactured by Invitrogen) containing 10% dFBS (manufactured by Invitrogen) and HT supplement (manufactured by Invitrogen) at 1.times. concentration) and the suspension was dispensed into a 96-well culture plate (manufactured by Iwaki Glass) at 100 .mu.l/well. The cells were cultured in a 5% CO.sub.2 incubator at 37.degree. C. for 24 hours, and then its medium was changed to IMDM-dFBS(10) (IMM medium containing 10% dialyzed FBS), followed by culturing for 1 to 2 weeks, Since colonies of transformants showing IT-independent growth were observed, the transformants in the wells in which growth was observed were subjected to a DHFR gene amplification, and the amount of the antibody production was increased. Specifically, the cells were suspended in IMDM-dFBS(10) medium containing 50 nM MTX at a density of 1 to 2.times.10.sup.5 cells/ml, and the suspension was dispensed to a 24-well plate (manufactured by Iwaki Glass) at 0.5 ml/well. The cells were cultured in a 5% CO.sub.2 incubator at 37.degree. C. for 1 to 2 weeks to induce transformants showing 50 r MTX resistance. Regarding the transformants in wells in which growth was observed, the MTX concentration of the medium was increased to 200 nM, and then a transformant capable of growing in the IMDM-dFBS(10) medium containing 200 nM MTX and of producing the anti-CD20 human chimeric antibody in a large amount was finally obtained in the same manner as described above.

Detail Description Paragraph - DETX (492):
[0643] The LCA Lectin-Resistant CHO/DG44 transformant cells capable of producing the anti-CD20 human chimeric antibody in a large amount obtained in the above item 2 was named R92-3-1. R92-3-1 has been deposited on Mar. 26, 2002, as FERM BP-7976 in International Patent Organism Depositary, National Insitute of Advanced Industrial Science and Technology (AIST Tsukuba Central 6, 1-1, Higashi 1-Chome Tsukaba-shi, Ibaraki-ken, Japan).

Detail Description Paragraph - DETX (495):
[0645] Purification of an Anti-CD20 Chimeric Antibody Produced by Lectin-Resistant CHO/DG44 Cell and Evaluation of its Activity

Detail Description Paragraph - DETX (496):
[0646] 1. Evaluation of Binding Activity of the Antibody Derived from Lectin-Resistant CHO/DG44 Cell (Immunofluorescent Method)

Detail Description Paragraph - DETX (498):
[0648] 2. Evaluation of In Vitro Cytotoxic Activity of the Antibody Derived from Lectin-Resistant CHO/DG44 Cell (ADCC Activity)

Detail Description Paragraph - DETX (500):
[0650] The results show that R92-3-1 antibody derived from LCA lectin-resistant CHO/DG44 Cell has Higher ADCC Activity than Rituxan.TM..

Detail Description Paragraph - DETX (501):
[0651] 3. Sugar Chain Analysis of the Antibody Derived from Lectin-Resistant CHO/DG44 Cell

Detail Description Paragraph - DETX (504):
[0654] As a result, in R92-3-1 antibody, the ratio of the .alpha.1,6-fucose-not-bound sugar chain group was 33%, whereas the ratio of the c1,6-fucose-bound sugar chains was 67%. When compared with the sugar chain analysis of Rituxan.TM. carried out in Example 3, the antibody produced by LCA lectin-resistant CHO/DC44 cells has a higher ratio of .alpha.1,6-fucose-not bound sugar chains.

Claims Text - CLTX (15):
14. The cell according to any one of claims 3 to 13, wherein the enzyme

activity is decreased or deleted by a technique selected from the group consisting of the following (a), (b), (c), (d) and (e): (a) a gene disruption technique targeting a gene encoding the enzyme; (b) a technique for introducing a dominant negative mutant of a gene encoding the enzyme; (c) a technique for introducing mutation into the enzyme; (d) a technique for inhibiting transcription or translation of a gene encoding the enzyme, (e) a technique for selecting a cell line resistant to a lectin which recognizes a sugar chain in which 1-position of fucose is bound to 6-position of-N-acetylglucosamine in the reducing end through .alpha.-bond in the complex N-glycoside-linked sugar chain.

Claims Text - CLTX (16):

15. The cell according to any one of claims 1 to 14, which is resistant to at least a lectin which recognizes a sugar chain in which 1-position of fucose is bound to 6-position of N-acetylglucosamine in the reducing end through .alpha.-bond in the complex N-glycoside-linked sugar chain.

PGPUB-DOCUMENT-NUMBER: 20040082009

PGPUB-FILING-TYPE: new

DOCUMENT-IDENTIFIER: US 20040082009 A1

TITLE: Compositions and methods for regulating receptor clustering

PUBLICATION-DATE: April 29, 2004

INVENTOR-INFORMATION:

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Demetriou, Michael	Irvine		US	

APPL-NO: 10/ 250935

DATE FILED: December 15, 2003

PCT-DATA:
APPL-NO: PCT/CA02/00002
DATE-FILED: Jan 11, 2002
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PUB-DATE:
371-DATE:
102(E)-DATE:

US-CL-CURRENT: 435/7.2, 530/370 , 530/395

ABSTRACT:

The invention relates to isolated complexes comprising one or more galectin associated with a Mgat5 modified glycan or polylactosamine modified glycan, and isolated lectin-Mgt5 modified glycan lattice comprising an array of mulitvalent interactions among lectins, Mgat5 modified glycans, polylactosamine modified glycans, and/or glycoproteins. Methods for evaluating a test compound for its ability to regulate receptor clustering through glycans on cell surfaces; and methods for regulating receptor clustering on cell surfaces comprising altering glycans on the cell surface associated with receptor clustering are also disclosed.

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Detail Description Paragraph - DETX (81):
[0107] In another method of the invention, immortalized cells expressing Mgat5-modified glycans may be treated with a substance suspected of inhibiting or stimulating Mgat5. The cells can be treated with a lectin such as L-PHA and the sensitivity to the lectin can be compared with controls cells which have not been treated with the substance and/or which do not express Mgat5. Examples of immortalized cells which can be used in the method are immortalized lung epithelial cell lines such as CHO cells, MvlLu cells, MDAY-D2 lymphoma, and lectin-resistant variants of these cell lines, which are transfected with a Mgat5 vector and MDCK cells. In the absence of an inhibitor the cells should show signs of morphologic transformation. In particular, morphologic transformation is evidenced by (a) fibroblastic morphology, spindle shape and

pile up; (b) the cells are less adhesive to substratum; (c) there is less cell-cell contact in monolayer culture; (d) there is reduced growth-factor requirements for survival and proliferation; (e) the cells grow in soft-agar or other semi-solid medium; (f) there is a lack of contact inhibition and increased apoptosis in low-serum high density cultures; (g) there is enhanced cell motility; and, (h) there is invasion into extracellular matrix and secretion of proteases. Substances which interfere with one or more of these phenotypes may be considered to inhibit Mgat5.

Detail Description Paragraph - DETX (152):

[0176] 15. Cummings, R. D., Trowbridge, I. S., and Kornfeld, S. A mouse lymphoma cell line resistant to the leukoagglutinating lectin from Phaseolus vulgaris is deficient in UDP-GlcNAc:.alpha.-D-mannoside .beta.1,6 N-acetylglucosaminyltransferase. J. Biol. Chem., 257: 13421-13427 (1982).

PGPUB-DOCUMENT-NUMBER: 20040029229

PGPUB-FILING-TYPE: new

DOCUMENT-IDENTIFIER: US 20040029229 A1

TITLE: High level protein expression system

PUBLICATION-DATE: February 12, 2004

INVENTOR-INFORMATION:

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Contreras, Roland	Merelbeke		BE	

APPL-NO: 10/ 441885

DATE FILED: May 20, 2003

RELATED-US-APPL-DATA:

non-provisional-of-provisional 60381978 20020520 US

US-CL-CURRENT: 435/69.1, 435/320.1 , 435/369

ABSTRACT:

The present application is directed to a system for expressing high levels of a protein of interest in a cell. Preferably the system includes introducing into a mammalian cell line an expression vector that comprises a gene encoding a selectable marker under the control of a weak promoter to facilitate integration of the expression vector into genomic areas that result in high levels of expression, along with a gene encoding the protein of interest. The gene of interest is linked to a strong promoter which results in high levels of expression of the desired protein. In one embodiment, the protein of interest is detrimental or toxic to cells, and the promoter is an inducible promoter. In another embodiment, the protein of interest is a glycoprotein, including membrane proteins, and the cell line has been mutated to inactivate N-acetylglucosamine transferase I to ensure uniform glycosylation. Preferably the cell is a mammalian or an insect cell.

CROSS-REFERENCE TO RELATED APPLICATIONS

[0001] This application claims the benefit of U.S. Provisional Application 60/381,978, filed May 20, 2002.

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Summary of Invention Paragraph - BSTX (27):
[0026] In one particularly preferred embodiment, the gene of interest encodes a glycoprotein, and the host cell line generates uniform glycosylation pattern on glycoproteins. Preferably, the host cell line is HEK293. One further preferred embodiment provides a ricin resistant cell line that is defective in GnTi activity.

Detail Description Paragraph - DETX (18):

[0063] To express a glycoprotein of interest with restricted or defined glycans, the cell line can been mutated to inactivate one of the enzymes involved in the oligosaccharide processing pathway in the ER and the Golgi apparatus, including the N-glycan biosynthesis paythway. For example, the cell line can be mutated to inactivate N-acetylglucosamine transferase I by selecting for mutants resistant to a lectin including RCA(II) (ricin). Glysoproteins expressed in such a mutant cell have unelaborated GlcNAc2Man5. Other useful lectins include PHA, WGA, ConA, and LCA. The enzyme activities in this pathway sensitive to this selection include alpha-3/6 mannosidase II, GlcNAc transferase II, core alpha6-fucosyltransferase, beta3 or 4 galactose transferase, beta3 or 4 GalNAc transferase, alpha2,3 and alpha 2,6-sialyltransferase.

Detail Description Paragraph - DETX (19):

[0064] For example, mutants deficient in N-acetylglucosamine transferase I, a key enzyme for complex N-glycan synthesis, can be isolated by selecting for ricin resistance. by treating exponentially growing cells with a mutagen and selecting for survivors in the presence of ricin. Mutagens include ethyl methane sulfonate and ultraviolet irradiation. Ricin can be used at 1 ng/ml, 10 ng/ml, 100 ng/ ml, or 1000 ng/ml. Cells can be refed with fresh medium containing ricin until colonies form. In a preferred embodiment, the cells are HEK293S cells.

Detail Description Paragraph - DETX (83):

[0125] Chemical Mutagenesis of HEK293S Cells and Isolation of Mutants Resistant to Ricin

Detail Description Paragraph - DETX (86):

[0128] Ricin.sup.R colonies were expanded to 10-cm dishes. Cells split 1:5 were transiently transfected the next day by using 30 .mu.g of a mixture (10:1 wt/wt) of two plasmids (pMT4 and pRSVTag, respectively) by the Ca.sub.2PO.sub.4 precipitation procedure (26). Plasmid pRSVTag (29) carries a gene encoding SV40 large tumor (T) antigen that promotes pMT4 replication in HEK293 S cells (29), whereas plasmid pMT4 carries the WT opsin gene (17). Transfection was performed as described (1) but after removal of the DNA mixture, cells were incubated for 48 h to allow transient expression of the opsin gene. Cells were harvested, treated with 11-cis-retinal (5 .mu.M), and solubilized by using buffer B (26) in the dark. Rhodopsin was purified by using mAb 1D4-Sepharose immunoaffinity chromatography (1, 17). Purified rhodopsin was subjected to SDS/PAGE (10%) and the bands were visualized by silver stain. The N-glycans inrhodopsin expressed transiently in the ricin-resistant cell lines were also analyzed by N-glycan-profiling experiments (see below).

Detail Description Paragraph - DETX (122):

[0164] In summary, these results show that an HEK293S cell line resistant to ricin was prepared by mutagenesis by using ethyl methanesulfonate. This cell line lacks N-acetylglucosaminyltransferase I (GnTI) activity, and consequently is unable to synthesize complex N-glycans. The tetracycline-inducible opsin expression system was assembled into this GnTI.sup.- HEK293S cell line. Stable cell lines were isolated that gave tetracycline/sodium butyrate-inducible expression of the WT opsin gene at levels comparable with those observed in the parent tetracycline-inducible HEK293S cell line. Analysis of the N-glycan in rhodopsin expressed by the HEK293S GnTI.sup.- stable cell line showed it to be Man.sub.5GlcNAc.sub.2. In a larger-scale expression experiment (1.1 liter) a WT opsin production level of 6 mg/liter was obtained. Further, the toxic constitutively active rhodopsin mutant, E113Q/E134Q/M257Y, previously shown to require inducible expression, has now been expressed in an HEK293S GNTI.sup.--inducible cell line at levels comparable with those obtained with WT

rhodopsin.

Claims Text - CLTX (19):
18. The cell line of claim 17, wherein the HEK293 cell line is a ricin resistant cell line that is defective in GnTi activity.

Claims Text - CLTX (27):
26. The method of claim 25, wherein the host cell line is a ricin resistant cell line that is defective in GnTi activity.

PGPUB-DOCUMENT-NUMBER: 20040002061

PGPUB-FILING-TYPE: new

DOCUMENT-IDENTIFIER: US 20040002061 A1

TITLE: Signal for packaging of influenza virus vectors

PUBLICATION-DATE: January 1, 2004

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
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APPL-NO: 10/ 366630

DATE FILED: February 12, 2003

RELATED-US-APPL-DATA:

non-provisional-of-provisional 60356538 20020213 US

non-provisional-of-provisional 60438679 20030107 US

US-CL-CURRENT: 435/5, 435/235.1 , 435/320.1 , 435/325 , 435/456 , 435/69.3

ABSTRACT:

The invention provides a packaging (incorporation) signal for influenza virus vectors, and methods of using the signal to transmit and maintain influenza viral and foreign nucleic acid in virus and cells.

CROSS-REFERENCE TO RELATED APPLICATIONS

[0001] This application claims the benefit of the filing date of U.S. Provisional Application Serial No. 60/356,538, filed on Feb. 13, 2002, and U.S. Provisional Application Serial No. _____, filed on Jan. 7, 2003, under 35 U.S.C. .sctn.119(e). The disclosure in those applications is incorporated by reference herein.

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Brief Description of Drawings Paragraph - DRTX (2):

[0022] FIG. 1. Binding of lectin-resistant cell lines. For each cell line, cells were incubated with digoxigenin-labeled Maakia amurensis (MAA) or Sambucus nigra (SNA) lectins, followed by fluorescein isothiocyanate-labeled antidigoxigenin antibody, and then analyzed by FACS. Bold lines, binding of the MAA lectin; narrow lines, binding of the SNA lectin; shaded profiles, negative control (no lectin added).

Detail Description Paragraph - DETX (55):

[0106] Generation of lectin-resistant cell lines. MDCK cells grown to 75% confluency were washed three times with phosphate-buffered saline and incubated with Maakia amurensis (MAA) lectin (100 mg/ml; Boehringer Mannheim, Mannheim, Germany) or Sambucus nigra (SNA) lectin (100 mg/ml; Boehringer Mannheim) in MEM containing 0.3% bovine serum albumin. After a 48 hour incubation, the medium

was replaced with growth medium (MEM-5% fetal calf serum). Lectin selection was repeated as above two additional times. Surviving cell colonies were then cloned, and the SNA-and MAA-selected cell lines were designated MDCK-Sn10 and MDCK-Ma, respectively.

Detail Description Paragraph - DETX (60):

[0111] Generation of lectin-resistant cell lines. To produce cell lines with a decreased level of sialic acid expression on the cell surface, two lectins were used, SNA and MAA, that differ in sialic acid-binding specificity. The MAA lectin binds to sialic acid linked to galactose by .alpha.(2,3) linkages (Wang et al., 1988), while the SNA lectin is specific for sialic acids linked to galactose or N-acetylgalactosamine by .alpha.(2-6) linkages (Shibuya et al., 1987). The MDCK cell line, which supports the growth of influenza viruses, was used as a parent cell for lectin selection. When incubated in the presence of either lectin, the majority of cells died within a week. Resistant cell clones were then grown out for stock cultures. The cell lines resulting from MAA and SNA lectin selection were designated MDCK-Ma and MDCK-SnO, respectively.

Detail Description Table CWU - DETL (1):

1TABLE 1 Replication of influenza viruses in lectin-resistant cell lines* Titer (TCID.sub.50/ml) Cell line AM2AL3 K4 MDCK 1.8 .times. 10.sup.9 5.6 .times. 10.sup.4 MDCK-Sn10 5.6 .times. 10.sup.8 3.2 .times. 10.sup.4 MDCK-Ma 1.8 .times. 10.sup.8 5.6 .times. 10.sup.3 *The susceptibility of each cell line was determined by infecting cells with AM2AL3 or K4 with virus and determining the dose required to infect 50% of tissue culture cells (TCID.sub.50).

PGPUB-DOCUMENT-NUMBER: 20030211075

PGPUB-FILING-TYPE: new

DOCUMENT-IDENTIFIER: US 20030211075 A1

TITLE: Combined compositions for tumor vasculature coagulation and treatment

PUBLICATION-DATE: November 13, 2003

INVENTOR-INFORMATION:

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King, Steven W.	Rancho Santa Margarita	CA	US	
Gottstein, Claudia	Dallas	TX	US	

APPL-NO: 10/ 259244

DATE FILED: September 27, 2002

RELATED-US-APPL-DATA:

non-provisional-of-provisional 60325532 20010927 US

US-CL-CURRENT: 424/85.1, 514/12 , 514/323 , 514/54 , 514/733

ABSTRACT:

Disclosed are various defined combinations of agents for use in improved anti-vascular therapies and coagulative tumor treatment. Particularly provided are combined treatment methods, and associated compositions, pharmaceuticals, medicaments, kits and uses, which together function surprisingly effectively in the treatment of vascularized tumors. The invention preferably involves a component or treatment step that enhances the effectiveness of therapy using targeted or non-targeted coagulants to cause tumor vasculature thrombosis.

[0001] Applicants claim priority to U.S. provisional application Serial No. 60/325,532, filed Sep. 27, 2001, the specification, claims and drawings of which application are specifically incorporated herein by reference without disclaimer.

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Detail Description Paragraph - DETX (665):

[0740] MHC Class II antigens are also expressed by B-lymphocytes, some bone marrow cells, myeloid cells and some renal and gut epithelia in BALB/c nu/nu mice, however, therapeutic doses of anti-Class II immunotoxin did not cause any permanent damage to these cell populations. Splenic B cells and bone marrow myelocytes bound intravenously injected anti-Class II antibody but early bone marrow progenitors do not express Class II antigens and mature bone marrow subsets and splenic B cell compartments were normal 3 weeks after therapy, so it is likely that any Ia.sup.+ myelocytes and B cells killed by the immunotoxin were replaced from the stem cell pool. It is contemplated that the existence of large numbers of readily accessible B cells in the spleen prevented the

anti-Class II immunotoxin from reaching the relatively inaccessible Ia.sup.+ epithelial cells but hepatic Kupffer cells were not apparently damaged by M5/114-dgA despite binding the immunotoxin. Myeloid cells are resistant to ricin A-chain immunotoxins, probably due to unique endocytic pathways related to their degradative physiologic function. No severe vascular-mediated toxicity was seen in the studies reported here because mice were maintained on oral antibiotics which minimized immune activity in the small intestine.

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PGPUB-FILING-TYPE: new

DOCUMENT-IDENTIFIER: US 20030185832 A1

TITLE: Combined methods and compositions for tumor vasculature targeting and tumor treatment

PUBLICATION-DATE: October 2, 2003

INVENTOR-INFORMATION:

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DATE FILED: February 27, 2003

RELATED-US-APPL-DATA:

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child 09738970 20001214 US

parent continuation-of 09207277 19981208 US GRANTED

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child 09207277 19981208 US

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parent-patent 5965132 US

child 08350212 19941205 US

parent continuation-in-part-of 08205330 19940302 US GRANTED

parent-patent 5855866 US

child 08205330 19940302 US

parent continuation-in-part-of 07846349 19920305 US ABANDONED

US-CL-CURRENT: 424/155.1, 424/145.1

ABSTRACT:

The present invention relates generally to methods and compositions for targeting the vasculature of solid tumors using immunological- and growth factor-based reagents. In particular aspects, antibodies carrying diagnostic or therapeutic agents are targeted to the vasculature of solid tumor masses through recognition of tumor vasculature-associated antigens, such as, for example, through endoglin binding, or through the specific induction of

endothelial cell surface antigens on vascular endothelial cells in solid tumors.

[0001] The present application is a continuation-in-part of co-pending U.S. patent application Ser. No. 08/205,330, filed Mar. 2, 1994; which is a continuation-in-part of U.S. patent application Ser. No. 07/846,349, filed Mar. 05, 1992. The entire text and figures of which disclosures are specifically incorporated by reference herein without disclaimer.

---------- KWIC ---------

Detail Description Paragraph - DETX (182):

[0248] MHC Class II antigens are also expressed by B-lymphocytes, some bone marrow cells, myeloid cells and some renal and gut epithelia in BALB/c nu/nu mice, however, therapeutic doses of anti-Class II immunotoxin did not cause any permanent damage to these cell populations. Splenic B cells and bone marrow myelocytes bound intravenously injected anti-Class II antibody but early bone marrow progenitors do not express Class II antigens and mature bone marrow subsets and splenic B cell compartments were normal 3 weeks after therapy, so it is likely that any Ia.sup.+ myelocytes and B cells killed by the immunotoxin were replaced from the stem cell pool. It is contemplated that the existence of large numbers of readily accessible B cells in the spleen prevented the anti-Class II immunotoxin from reaching the relatively inaccessible Ia.sup.+ epithelial cells but hepatic Kupffer cells were not apparently damaged by M5/114-dgA despite binding the immunotoxin. Myeloid cells are resistant to ricin A-chain immunotoxins, probably due to unique endocytic pathways related to their degradative physiologic function (Engert et al., 1991).

US-PAT-NO: 6710228

DOCUMENT-IDENTIFIER: US 6710228 B1

TITLE: Cotton cells, plants, and seeds genetically engineered to express insecticidal and fungicidal chitin binding proteins (lectins)

DATE-ISSUED: March 23, 2004

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Yenofsky; Richard L.	Arcadia	CA	N/A	N/A
Fine; Miriam	Arcadia	CA	N/A	N/A
Rangan; Thirumale S.	Lubbock	TX	N/A	N/A
Anderson; David M.	Placentia	CA	N/A	N/A

APPL-NO: 09/ 322640

DATE FILED: May 28, 1999

PARENT-CASE:

CROSS-REFERENCE TO A RELATED APPLICATION

This application claims the benefit of provisional application Serial No. 60/087,219; filed May 29, 1998, which is hereby incorporated by reference in its entirety.

US-CL-CURRENT: 800/279, 435/419 , 435/468 , 435/69.1 , 536/23.1 , 536/23.6 , 800/278 , 800/295 , 800/298 , 800/301 , 800/302 , 800/314

ABSTRACT:

Chimeric genes encoding lectins exhibiting pesticidal activity (for example, insecticidal and/or fungicidal activity) are disclosed which can be used to transform cotton to yield cotton cells, plants, and seeds in which the chimeric genes are expressed. Such transformed cotton cells are pesticidal when ingested by cotton pests.

21 Claims, 4 Drawing figures

Exemplary Claim Number: 1

Number of Drawing Sheets: 4

--------- KWIC --------

Other Reference Publication - OREF (18):
Rajguru, S. et al. "Assessment of Resistance of Cotton Transformed with Lectin Genes to Tobacco Budworm" 1998 Proceedings Beltwide Cotton Conferences, San Diego, California, USA Jan. 5-9, 1998. vol. I, (1998) PP490-491, Jan. 1998.

Other Reference Publication - OREF (19):
Rajguru, S., et al. "Assessment of resistance of cotton transformed with lectin genes to tobacco budworm" Special Report--Arkansas Agricultural

Experiment Station 1998 No. 188 pp. 95-98 1998.

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  * * * * * * * * * * * * * STN  Columbus  * * * * * * * * * * * * * *

FILE 'HOME' ENTERED AT 12:56:05 ON 01 JUN 2004

=> fil .bec
COST IN U.S. DOLLARS                                  SINCE FILE      TOTAL
                                                         ENTRY    SESSION
FULL ESTIMATED COST                                       0.21       0.21

FILES 'MEDLINE, SCISEARCH, LIFESCI, BIOTECHDS, BIOSIS, EMBASE, HCAPLUS, NTIS,
       ESBIOBASE, BIOTECHNO, WPIDS' ENTERED AT 12:56:17 ON 01 JUN 2004
ALL COPYRIGHTS AND RESTRICTIONS APPLY.  SEE HELP USAGETERMS FOR DETAILS.

11 FILES IN THE FILE LIST

=> s (lectin# or hemagglutin? or ricin)(2a)resist?
FILE 'MEDLINE'
         37257 LECTIN#
         47141 HEMAGGLUTIN?
          2612 RICIN
        405664 RESIST?
L1         445 (LECTIN# OR HEMAGGLUTIN? OR RICIN)(2A)RESIST?

FILE 'SCISEARCH'
         26124 LECTIN#
         13775 HEMAGGLUTIN?
          2914 RICIN
        507953 RESIST?
L2         355 (LECTIN# OR HEMAGGLUTIN? OR RICIN)(2A)RESIST?

FILE 'LIFESCI'
          8887 LECTIN#
          8410 HEMAGGLUTIN?
           966 RICIN
        126192 RESIST?
L3         210 (LECTIN# OR HEMAGGLUTIN? OR RICIN)(2A)RESIST?

FILE 'BIOTECHDS'
          1117 LECTIN#
          1176 HEMAGGLUTIN?
           339 RICIN
         29109 RESIST?
L4          24 (LECTIN# OR HEMAGGLUTIN? OR RICIN)(2A)RESIST?

FILE 'BIOSIS'
         32677 LECTIN#
         21393 HEMAGGLUTIN?
          3014 RICIN
        462212 RESIST?
L5         659 (LECTIN# OR HEMAGGLUTIN? OR RICIN)(2A)RESIST?

FILE 'EMBASE'
         23005 LECTIN#
         18690 HEMAGGLUTIN?
          2493 RICIN
        363435 RESIST?
L6         377 (LECTIN# OR HEMAGGLUTIN? OR RICIN)(2A)RESIST?

FILE 'HCAPLUS'
         36038 LECTIN#
         23388 HEMAGGLUTIN?
          3233 RICIN
       1380813 RESIST?
L7         584 (LECTIN# OR HEMAGGLUTIN? OR RICIN)(2A)RESIST?
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FILE 'NTIS'
           140 LECTIN#
           665 HEMAGGLUTIN?
            91 RICIN
         70936 RESIST?
L8           3 (LECTIN# OR HEMAGGLUTIN? OR RICIN)(2A)RESIST?

FILE 'ESBIOBASE'
          8314 LECTIN#
          3592 HEMAGGLUTIN?
           675 RICIN
        121782 RESIST?
L9           76 (LECTIN# OR HEMAGGLUTIN? OR RICIN)(2A)RESIST?

FILE 'BIOTECHNO'
          9786 LECTIN#
          8088 HEMAGGLUTIN?
          1182 RICIN
        103753 RESIST?
L10         263 (LECTIN# OR HEMAGGLUTIN? OR RICIN)(2A)RESIST?

FILE 'WPIDS'
          2036 LECTIN#
           480 HEMAGGLUTIN?
           574 RICIN
       1005637 RESIST?
L11          12 (LECTIN# OR HEMAGGLUTIN? OR RICIN)(2A)RESIST?

TOTAL FOR ALL FILES
L12        3008 (LECTIN# OR HEMAGGLUTIN? OR RICIN)(2A) RESIST?

=> s l12 and lysosom?
FILE 'MEDLINE'
         39021 LYSOSOM?
L13          4 L1 AND LYSOSOM?

FILE 'SCISEARCH'
         22909 LYSOSOM?
L14          1 L2 AND LYSOSOM?

FILE 'LIFESCI'
          7194 LYSOSOM?
L15          0 L3 AND LYSOSOM?

FILE 'BIOTECHDS'
           488 LYSOSOM?
L16          2 L4 AND LYSOSOM?

FILE 'BIOSIS'
         38506 LYSOSOM?
L17          4 L5 AND LYSOSOM?

FILE 'EMBASE'
         30316 LYSOSOM?
L18          3 L6 AND LYSOSOM?

FILE 'HCAPLUS'
         33872 LYSOSOM?
L19          6 L7 AND LYSOSOM?

FILE 'NTIS'
           280 LYSOSOM?
L20          0 L8 AND LYSOSOM?
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FILE 'ESBIOBASE'
          8550 LYSOSOM?
L21          0 L9 AND LYSOSOM?

FILE 'BIOTECHNO'
          8722 LYSOSOM?
L22          1 L10 AND LYSOSOM?

FILE 'WPIDS'
           600 LYSOSOM?
L23          2 L11 AND LYSOSOM?

TOTAL FOR ALL FILES
L24         23 L12 AND LYSOSOM?

=> s l12 and mannose
FILE 'MEDLINE'
         17284 MANNOSE
L25        222 L1 AND MANNOSE

FILE 'SCISEARCH'
         12985 MANNOSE
L26        158 L2 AND MANNOSE

FILE 'LIFESCI'
          5734 MANNOSE
L27        133 L3 AND MANNOSE

FILE 'BIOTECHDS'
          1672 MANNOSE
L28         12 L4 AND MANNOSE

FILE 'BIOSIS'
         19914 MANNOSE
L29        373 L5 AND MANNOSE

FILE 'EMBASE'
         13513 MANNOSE
L30        177 L6 AND MANNOSE

FILE 'HCAPLUS'
         35664 MANNOSE
L31        242 L7 AND MANNOSE

FILE 'NTIS'
           112 MANNOSE
L32          1 L8 AND MANNOSE

FILE 'ESBIOBASE'
          5153 MANNOSE
L33         35 L9 AND MANNOSE

FILE 'BIOTECHNO'
          7168 MANNOSE
L34        151 L10 AND MANNOSE

FILE 'WPIDS'
          2583 MANNOSE
L35          3 L11 AND MANNOSE

TOTAL FOR ALL FILES
L36       1507 L12 AND MANNOSE
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=> s l12 and high(2a)mannose
FILE 'MEDLINE'
       1212315 HIGH
         17284 MANNOSE
          2004 HIGH(2A)MANNOSE
L37         11 L1 AND HIGH(2A)MANNOSE

FILE 'SCISEARCH'
       1767795 HIGH
         12985 MANNOSE
          1332 HIGH(2A)MANNOSE
L38          2 L2 AND HIGH(2A)MANNOSE

FILE 'LIFESCI'
        333161 HIGH
          5734 MANNOSE
           659 HIGH(2A)MANNOSE
L39          1 L3 AND HIGH(2A)MANNOSE

FILE 'BIOTECHDS'
         62833 HIGH
          1672 MANNOSE
           144 HIGH(2A)MANNOSE
L40          2 L4 AND HIGH(2A)MANNOSE

FILE 'BIOSIS'
       1349013 HIGH
         19914 MANNOSE
          2153 HIGH(2A)MANNOSE
L41         10 L5 AND HIGH(2A)MANNOSE

FILE 'EMBASE'
       1168335 HIGH
         13513 MANNOSE
          1661 HIGH(2A)MANNOSE
L42         10 L6 AND HIGH(2A)MANNOSE

FILE 'HCAPLUS'
       3362832 HIGH
         35664 MANNOSE
          2460 HIGH(2A)MANNOSE
L43         15 L7 AND HIGH(2A)MANNOSE

FILE 'NTIS'
        319287 HIGH
           112 MANNOSE
             6 HIGH(2A)MANNOSE
L44          0 L8 AND HIGH(2A)MANNOSE

FILE 'ESBIOBASE'
        413248 HIGH
          5153 MANNOSE
           645 HIGH(2A)MANNOSE
L45          0 L9 AND HIGH(2A)MANNOSE

FILE 'BIOTECHNO'
        299126 HIGH
          7168 MANNOSE
          1224 HIGH(2A)MANNOSE
L46          9 L10 AND HIGH(2A)MANNOSE

FILE 'WPIDS'
       1831128 HIGH
          2583 MANNOSE
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```
            72 HIGH(2A)MANNOSE
L47          2 L11 AND HIGH(2A)MANNOSE

TOTAL FOR ALL FILES
L48         62 L12 AND HIGH(2A) MANNOSE

=> s l36 and recombinant?
FILE 'MEDLINE'
        227075 RECOMBINANT?
L49         15 L25 AND RECOMBINANT?

FILE 'SCISEARCH'
        135705 RECOMBINANT?
L50          5 L26 AND RECOMBINANT?

FILE 'LIFESCI'
         62786 RECOMBINANT?
L51          5 L27 AND RECOMBINANT?

FILE 'BIOTECHDS'
         82006 RECOMBINANT?
L52          4 L28 AND RECOMBINANT?

FILE 'BIOSIS'
        178230 RECOMBINANT?
L53         19 L29 AND RECOMBINANT?

FILE 'EMBASE'
        149031 RECOMBINANT?
L54         12 L30 AND RECOMBINANT?

FILE 'HCAPLUS'
        161309 RECOMBINANT?
L55         14 L31 AND RECOMBINANT?

FILE 'NTIS'
          1557 RECOMBINANT?
L56          0 L32 AND RECOMBINANT?

FILE 'ESBIOBASE'
         72176 RECOMBINANT?
L57          3 L33 AND RECOMBINANT?

FILE 'BIOTECHNO'
        127206 RECOMBINANT?
L58         12 L34 AND RECOMBINANT?

FILE 'WPIDS'
         35637 RECOMBINANT?
L59          0 L35 AND RECOMBINANT?

TOTAL FOR ALL FILES
L60         89 L36 AND RECOMBINANT?

=> s l24 or l48 or l60
FILE 'MEDLINE'
L61         30 L13 OR L37 OR L49

FILE 'SCISEARCH'
L62          8 L14 OR L38 OR L50

FILE 'LIFESCI'
L63          6 L15 OR L39 OR L51
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FILE 'BIOTECHDS'
L64 5 L16 OR L40 OR L52

FILE 'BIOSIS'
L65 33 L17 OR L41 OR L53

FILE 'EMBASE'
L66 25 L18 OR L42 OR L54

FILE 'HCAPLUS'
L67 35 L19 OR L43 OR L55

FILE 'NTIS'
L68 0 L20 OR L44 OR L56

FILE 'ESBIOBASE'
L69 3 L21 OR L45 OR L57

FILE 'BIOTECHNO'
L70 22 L22 OR L46 OR L58

FILE 'WPIDS'
L71 2 L23 OR L47 OR L59

TOTAL FOR ALL FILES
L72 169 L24 OR L48 OR L60

=> dup rem l72
PROCESSING COMPLETED FOR L72
L73 56 DUP REM L72 (113 DUPLICATES REMOVED)

=> d tot

L73 ANSWER 1 OF 56 BIOTECHDS COPYRIGHT 2004 THOMSON DERWENT/ISI on STN
TI Producing a glycoprotein with reduced complex carbohydrates by culturing the **lectin resistant** mammalian cell expressing the glycoprotein for treating **lysosomal** storage disease; **recombinant** protein production via host cell culture for use in disease therapy and gene therapy
AU CANFIELD W M
AN 2004-00315 BIOTECHDS
PI US 2003124653 3 Jul 2003

L73 ANSWER 2 OF 56 BIOTECHDS COPYRIGHT 2004 THOMSON DERWENT/ISI on STN
TI Producing a **high mannose** glycoprotein for treating **lysosomal** storage disease, comprises culturing the **lectin resistant** mammalian cell in the presence of deoxymannojirimycin and kifunensine; protein production via host cell culture for use in disease therapy and gene therapy
AU CANFIELD W M
AN 2004-00314 BIOTECHDS
PI US 2003124652 3 Jul 2003

L73 ANSWER 3 OF 56 HCAPLUS COPYRIGHT 2004 ACS on STN
TI Potent anti-influenza activity of cyanovirin-N and interactions with viral hemagglutinin
SO Antimicrobial Agents and Chemotherapy (2003), 47(8), 2518-2525
CODEN: AMACCQ; ISSN: 0066-4804
AU O'Keefe, Barry R.; Smee, Donald F.; Turpin, Jim A.; Saucedo, Carrie J.; Gustafson, Kirk R.; Mori, Toshiyuki; Blakeslee, Dennis; Buckheit, Robert; Boyd, Michael R.
AN 2003:594600 HCAPLUS
DN 140:52807

L73 ANSWER 4 OF 56 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
TI Identification of mrpI as the only recombinase that regulates the phase-variable expression of the MR/P fimbriae of uropathogenic Proteus mirabilis.
SO Abstracts of the General Meeting of the American Society for Microbiology, (2001) Vol. 101, pp. 60. print.
Meeting Info.: 101st General Meeting of the American Society for Microbiology. Orlando, FL, USA. May 20-24, 2001. American Society for Microbiology.
ISSN: 1060-2011.
AU Li, X. [Reprint author]; Lockatell, V. [Reprint author]; Johnson, D. E.; Mobley, H. L. T. [Reprint author]
AN 2002:176365 BIOSIS

L73 ANSWER 5 OF 56 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
TI Molecular characterization of papA gene from uropathogenic Escherichia coli.
SO Journal of the Korean Society for Microbiology, (August, 1999) Vol. 34, No. 4, pp. 385-392. print.
CODEN: TMHCDX. ISSN: 0253-3162.
AU Song, Hye-Wone [Reprint author]; Kim, Jong-Bae [Reprint author]
AN 2000:360700 BIOSIS

L73 ANSWER 6 OF 56 MEDLINE on STN DUPLICATE 3
TI New fimbrial adhesins of Serratia marcescens isolated from urinary tract infections: description and properties.
SO Journal of urology, (1997 Feb) 157 (2) 694-8.
Journal code: 0376374. ISSN: 0022-5347.
AU Leranoz S; Orus P; Berlanga M; Dalet F; Vinas M
AN 97149592 MEDLINE

L73 ANSWER 7 OF 56 Elsevier BIOBASE COPYRIGHT 2004 Elsevier Science B.V. on STN
AN 1998029381 ESBIOBASE
TI Adherence pili of pathogenic strains of avian Escherichia coli
AU Vidotto M.C.; Navarro H.R.; Gaziri L.C.J.
CS M.C. Vidotto, Universidade Estadual de Londrina, Depto de Patologia Geral, Campus Universitario, Caixa postal 6001, 86051-970 Londrina, Brazil.
E-mail: vidotto@npd.uel.br
SO Veterinary Microbiology, (1997), 59/1 (79-87), 29 reference(s)
CODEN: VMICDQ ISSN: 0378-1135
PUI S0378113597001247
DT Journal; Article
CY Netherlands
LA English
SL English

L73 ANSWER 8 OF 56 MEDLINE on STN DUPLICATE 4
TI Preparation of antisera to **recombinant**, soluble N-acetylglucosaminyltransferase V and its visualization in situ.
SO Glycoconjugate journal, (1995 Dec) 12 (6) 813-23.
Journal code: 8603310. ISSN: 0282-0080.
AU Chen L; Zhang N; Adler B; Browne J; Freigen N; Pierce M
AN 96318020 MEDLINE

L73 ANSWER 9 OF 56 MEDLINE on STN DUPLICATE 5
TI Cloning, sequencing, and viscometric adhesion analysis of heat-resistant agglutinin 1, an integral membrane hemagglutinin from Escherichia coli O9:H10:K99.
SO Infection and immunity, (1994 Nov) 62 (11) 5020-6.
Journal code: 0246127. ISSN: 0019-9567.
AU Lutwyche P; Rupps R; Cavanagh J; Warren R A; Brooks D E

AN 95012721 MEDLINE

L73 ANSWER 10 OF 56 MEDLINE on STN DUPLICATE 6
TI Increased LAMP-2 polylactosamine glycosylation is associated with its slower Golgi transit during establishment of a polarized MDCK epithelial monolayer.
SO Molecular biology of the cell, (1993 Jun) 4 (6) 627-35.
Journal code: 9201390. ISSN: 1059-1524.
AU Nabi I R; Rodriguez-Boulan E
AN 93385651 MEDLINE

L73 ANSWER 11 OF 56 HCAPLUS COPYRIGHT 2004 ACS on STN
TI Ricin-mediated cell lysis and apoptosis of drug sensitive and resistant tumor cells
SO International Journal of Oncology (1993), 2(3), 363-71
CODEN: IJONES; ISSN: 1019-6439
AU Morimoto, Hideki; Bonavida, Benjamin
AN 1993:400387 HCAPLUS
DN 119:387

L73 ANSWER 12 OF 56 HCAPLUS COPYRIGHT 2004 ACS on STN
TI Purification and properties of **recombinant** K99 and F41 fimbriae antigens from engineered strain
SO Shengwu Huaxue Zazhi (1992), 8(2), 190-4
CODEN: SHZAE4; ISSN: 1000-8543
AU Zhong, Sheng; Li, Fengsheng; Rui, Xianliang; Lu, Xuexian; Wang, Qingyuan; Huang, Peitang
AN 1992:424415 HCAPLUS
DN 117:24415

L73 ANSWER 13 OF 56 MEDLINE on STN DUPLICATE 7
TI Molecular cloning, expression, and sequence of the pilin gene from nontypeable Haemophilus influenzae M37.
SO Infection and immunity, (1991 May) 59 (5) 1716-22.
Journal code: 0246127. ISSN: 0019-9567.
AU Coleman T; Grass S; Munson R Jr
AN 91209926 MEDLINE

L73 ANSWER 14 OF 56 SCISEARCH COPYRIGHT 2004 THOMSON ISI on STN
TI ALPHA-HEMOLYSIN CONTRIBUTES TO THE PATHOGENICITY OF PILIATED DIGALACTOSIDE-BINDING ESCHERICHIA-COLI IN THE KIDNEY - EFFICACY OF AN ALPHA-HEMOLYSIN VACCINE IN PREVENTING RENAL INJURY IN THE BALB/C MOUSE MODEL OF PYELONEPHRITIS
SO INFECTION AND IMMUNITY, (1991) Vol. 59, No. 3, pp. 1153-1161.
AU OHANLEY P (Reprint); LALONDE G; JI G
AN 91:128718 SCISEARCH

L73 ANSWER 15 OF 56 SCISEARCH COPYRIGHT 2004 THOMSON ISI on STN
TI CHAPERONE-ASSISTED ASSEMBLY AND MOLECULAR ARCHITECTURE OF ADHESIVE PILI
SO ANNUAL REVIEW OF MICROBIOLOGY, (1991) Vol. 45, pp. 383-415.
AU HULTGREN S J (Reprint); NORMARK S; ABRAHAM S N
AN 91:557663 SCISEARCH

L73 ANSWER 16 OF 56 MEDLINE on STN
TI Molecular structure of the Dr adhesin: nucleotide sequence and mapping of receptor-binding domain by use of fusion constructs.
SO Infection and immunity, (1991 Jan) 59 (1) 261-8.
Journal code: 0246127. ISSN: 0019-9567.
AU Swanson T N; Bilge S S; Nowicki B; Moseley S L
AN 91099972 MEDLINE

L73 ANSWER 17 OF 56 SCISEARCH COPYRIGHT 2004 THOMSON ISI on STN
TI EFFECTS OF BREFELDIN-A ON OLIGOSACCHARIDE PROCESSING - EVIDENCE FOR DECREASED BRANCHING OF COMPLEX-TYPE GLYCANS AND INCREASED FORMATION OF

HYBRID-TYPE GLYCANS
SO BIOCHEMICAL JOURNAL, (1991) Vol. 279, No. OCT, pp. 159-165.
AU CHAWLA D; HUGHES R C (Reprint)
AN 91:565141 SCISEARCH

L73 ANSWER 18 OF 56 HCAPLUS COPYRIGHT 2004 ACS on STN
TI Enterotoxins, colonization factors and serotypes of enterotoxigenic Escherichia coli from humans and animals
SO Microbiologia (Madrid) (1991), 7(2), 57-73
CODEN: MICBE3; ISSN: 0213-4101
AU Blanco, Jorge; Blanco, Miguel; Garabal, Jose I.; Gonzalez, Enrique A.
AN 1992:169708 HCAPLUS
DN 116:169708

L73 ANSWER 19 OF 56 MEDLINE on STN DUPLICATE 8
TI Bovine and mouse serum beta inhibitors of influenza A viruses are mannose-binding lectins.
SO Proceedings of the National Academy of Sciences of the United States of America, (1990 Jun) 87 (12) 4485-9.
Journal code: 7505876. ISSN: 0027-8424.
AU Anders E M; Hartley C A; Jackson D C
AN 90280400 MEDLINE

L73 ANSWER 20 OF 56 MEDLINE on STN
TI Pyelonephritogenic Escherichia coli and killing of cultured human renal proximal tubular epithelial cells: role of hemolysin in some strains.
SO Infection and immunity, (1990 May) 58 (5) 1281-9.
Journal code: 0246127. ISSN: 0019-9567.
AU Mobley H L; Green D M; Trifillis A L; Johnson D E; Chippendale G R; Lockatell C V; Jones B D; Warren J W
AN 90215993 MEDLINE

L73 ANSWER 21 OF 56 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN DUPLICATE 9
TI HELA CELL MUTANTS RESISTANT TO EPIDERMAL GROWTH FACTOR RICIN A-CHAIN CONJUGATE.
SO Journal of Cellular Physiology, (1989) Vol. 139, No. 1, pp. 42-50.
CODEN: JCLLAX. ISSN: 0021-9541.
AU BANKER D [Reprint author]; HERSCHMAN H R
AN 1989:296021 BIOSIS

L73 ANSWER 22 OF 56 MEDLINE on STN DUPLICATE 10
TI Escherichia coli F41 adhesin: genetic organization, nucleotide sequence, and homology with the K88 determinant.
SO Journal of bacteriology, (1988 Oct) 170 (10) 4890-6.
Journal code: 2985120R. ISSN: 0021-9193.
AU Anderson D G; Moseley S L
AN 89008112 MEDLINE

L73 ANSWER 23 OF 56 MEDLINE on STN DUPLICATE 11
TI Distribution and degree of heterogeneity of the afimbrial-adhesin-encoding operon (afa) among uropathogenic Escherichia coli isolates.
SO Infection and immunity, (1988 Mar) 56 (3) 640-8.
Journal code: 0246127. ISSN: 0019-9567.
AU Labigne-Roussel A; Falkow S
AN 88138463 MEDLINE

L73 ANSWER 24 OF 56 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
TI STUDIES ON CLONING OF K-99 PILUS GENE OF ENTEROTOXIGENIC ESCHERICHIA-COLI ISOLATED FROM CALVES WITH DIARRHEA.
SO Research Reports of the Rural Development Administration (Suweon), (1988) Vol. 30, No. 1 VET, pp. 1-13.
ISSN: 1013-9397.
AU YOON Y D [Reprint author]; KIM J M; KO G W; JOO Y S; PARK J M

AN 1989:125917 BIOSIS

L73 ANSWER 25 OF 56 MEDLINE on STN DUPLICATE 12
TI Molecular cloning of the Escherichia coli O75X adhesin.
SO Infection and immunity, (1987 Dec) 55 (12) 3168-73.
Journal code: 0246127. ISSN: 0019-9567.
AU Nowicki B; Barrish J P; Korhonen T; Hull R A; Hull S I
AN 88057614 MEDLINE

L73 ANSWER 26 OF 56 MEDLINE on STN DUPLICATE 13
TI Characterization of a mannosidase acting on alpha 1----3- and alpha 1----6-linked mannose residues of oligomannosidic intermediates of glycoprotein processing.
SO European journal of biochemistry / FEBS, (1987 Oct 15) 168 (2) 287-94.
Journal code: 0107600. ISSN: 0014-2956.
AU Monis E; Bonay P; Hughes R C
AN 88029441 MEDLINE

L73 ANSWER 27 OF 56 MEDLINE on STN DUPLICATE 14
TI Role of Escherichia coli alpha-hemolysin and bacterial adherence in infection: requirement for release of inflammatory mediators from granulocytes and mast cells.
SO Infection and immunity, (1986 Dec) 54 (3) 886-92.
Journal code: 0246127. ISSN: 0019-9567.
AU Konig B; Konig W; Scheffer J; Hacker J; Goebel W
AN 87056094 MEDLINE

L73 ANSWER 28 OF 56 MEDLINE on STN
TI Cloning of chromosomal DNA encoding the F41 adhesin of enterotoxigenic Escherichia coli and genetic homology between adhesins F41 and K88.
SO Journal of bacteriology, (1986 Sep) 167 (3) 799-804.
Journal code: 2985120R. ISSN: 0021-9193.
AU Moseley S L; Dougan G; Schneider R A; Moon H W
AN 86304168 MEDLINE

L73 ANSWER 29 OF 56 MEDLINE on STN DUPLICATE 15
TI Contribution of cloned virulence factors from uropathogenic Escherichia coli strains to nephropathogenicity in an experimental rat pyelonephritis model.
SO Infection and immunity, (1986 Dec) 54 (3) 761-7.
Journal code: 0246127. ISSN: 0019-9567.
AU Marre R; Hacker J; Henkel W; Goebel W
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L73 ANSWER 30 OF 56 MEDLINE on STN DUPLICATE 16
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L73 ANSWER 53 OF 56 MEDLINE on STN DUPLICATE 31
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L73 ANSWER 56 OF 56 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
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CODEN: JGMIAN. ISSN: 0022-1287.
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L73 ANSWER 1 OF 56 BIOTECHDS COPYRIGHT 2004 THOMSON DERWENT/ISI on STN
AB DERWENT ABSTRACT:
NOVELTY - Producing a glycoprotein with reduced complex carbohydrates comprises: (a) introducing and expressing a polynucleotide encoding a glycoprotein into a mammalian cell; (b) culturing the mammalian cell in the presence of a lectin to obtain a **lectin resistant** mammalian cell; (c) isolating the **lectin resistant** mammalian cell; (d) culturing the **lectin resistant** mammalian cell expressing the glycoprotein; and (e) collecting the glycoprotein from the **lectin resistant** cells.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following: (1) treating a patient suffering from a **lysosomal** storage disease; and (2) making a mammalian cell that produces glycoproteins with reduced complex carbohydrates.

BIOTECHNOLOGY - Preferred Method: Producing a glycoprotein with reduced complex carbohydrates comprises: (a) introducing and expressing a polynucleotide encoding a glycoprotein into a mammalian cell; (b) selecting a mammalian cell expressing the glycoprotein that is **resistant** to a **lectin**; (c) culturing the **lectin resistant** mammalian cell; and (d) collecting the glycoprotein from the **lectin resistant** cells. The **lectin** comprises concanavalin A, erthroglutinin, lymphoagglutanin, wheat germ agglutinin, or preferably ricin. The glycoprotein is a **lysosomal** hydrolase, which is acid alpha-glucosidase. The GlcNAc-phosphotransferase comprises a 1199, 928, 328 or 305 residue amino acid sequence, given in the specification. It is encoded by: (a) a 3600 base pair sequence (S1), given in the specification; or (b) a sequence that hybridizes under

stringent conditions to the complement of (a). The GlcNAc-phosphotransferase is also encoded by: (a) (S1); or (b) a sequence that hybridizes under stringent conditions to the complement of (a). The GlcNAc-phosphotransferase comprises alpha-subunit and beta-subunit, which are encoded by: (a) a 5597 base pair sequence, given in the specification; or (b) a sequence that hybridizes under stringent conditions to the complement of (a). The GlcNAc-phosphotransferase also comprises a gamma subunit, which is encoded by: (a) a 1219 base pair sequence, given in the specification; or (b) a sequence that hybridizes under stringent conditions to the complement of (a). The deoxymannojirimycin comprises 0.1-5 mM. The kifunensine comprises 0.1-10 microg/ml. The phosphodiester alpha-GlcNAcase comprises a 515 amino acid sequence, given in the specification. It is encoded by: (a) a 2183 base pair sequence, given in the specification; or (b) a sequence that hybridizes under stringent conditions to the complement of (a). Treating a patient suffering from a **lysosomal** storage disease comprises: (a) culturing a **lectin resistant** mammalian cell in the presence of deoxymannojirimycin and kifunensine to inhibit glycosylation of the glycoprotein; (b) collecting the **high mannose** glycoprotein; (c) collecting the **lysosomal** hydrolase from the **lectin resistant** cells; (d) contacting the collected **lysosomal** hydrolase with a GlcNAc-phosphotransferase; and (e) contacting the **lysosomal** hydrolase with a phosphodiester alpha GlcNAcase after the contacting with a GlcNAc-phosphotransferase. The method further comprises: (a) transferring a N-acetylglucosamine-1-phosphate from UDP-GlcNAc to the glycoprotein; (b) purifying the glycoprotein comprising N-acetylglucosamine-1-phosphate; and (c) removing a N-acetylglucosamine from the glycoprotein. Making a mammalian cell that produces glycoproteins with reduced complex carbohydrates comprises: (a) introducing and expressing a polynucleotide encoding a glycoprotein into a mammalian cell; (b) culturing the mammalian cell in the presence of a lectin to obtain a **lectin resistant** mammalian cell; and (c) isolating the **lectin resistant** mammalian cell.

ACTIVITY - Gastrointestinal. No biological data given.

MECHANISM OF ACTION - Gene therapy.

USE - The method is useful for producing a glycoprotein with reduced complex carbohydrates (claimed) for treating **lysosomal** storage disease. (46 pages)

L73 ANSWER 2 OF 56 BIOTECHDS COPYRIGHT 2004 THOMSON DERWENT/ISI on STN

AB DERWENT ABSTRACT:

NOVELTY - Producing a **high mannose** glycoprotein comprises: (a) introducing and expressing a polynucleotide encoding a glycoprotein into a mammalian cell; (b) culturing the cell in the presence of a lectin to obtain a **lectin resistant** cell; (c) isolating the cell; (d) culturing the cell in the presence of deoxymannojirimycin and kifunensine to inhibit glycosylation of the glycoprotein; and (e) collecting the glycoprotein.

DETAILED DESCRIPTION - Producing a **high mannose** glycoprotein comprises: (a) introducing and expressing a polynucleotide encoding a glycoprotein into a mammalian cell; (b) culturing the mammalian cell in the presence of a lectin to obtain a **lectin resistant** mammalian cell; (c) isolating the **lectin resistant** mammalian cell; (d) culturing the **lectin resistant** mammalian cell in the presence of deoxymannojirimycin and kifunensine to inhibit glycosylation of the glycoprotein; and (e) collecting the **high mannose** glycoprotein. An INDEPENDENT CLAIM is also included for a method of treating a patient suffering from a **lysosomal** storage disease.

BIOTECHNOLOGY - Preferred Method: Producing a **high mannose** glycoprotein further comprises: (1) contacting the collected glycoprotein with a GlcNAc-phosphotransferase and with a

phosphodiester alpha-GlcNAcase; and (2) purifying the glycoprotein after the contacting. The lectin comprises concanavalin A, erthroglutinin, lymphoagglutanin, wheat germ agglutinin, or preferably ricin. The glycoprotein is a **lysosomal** hydrolase, which is acid alpha-glucosidase. The GlcNAc-phosphotransferase comprises a sequence having 1199, 928, 328 or 305 amino acids. It is encoded by: (1) a sequence comprising 3600 bp; or (2) a sequence that hybridizes under stringent conditions to the complement of (1). The GlcNAc-phosphotransferase is also encoded by: (1) a sequence comprising 3600 bp; or (2) a sequence that hybridizes under stringent conditions to the complement of (1). The GlcNAc-phosphotransferase comprises alpha-subunit and beta-subunit, which are encoded by: (1) a sequence having 5597 bp; or (2) a sequence that hybridizes under stringent conditions to the complement of (1). The GlcNAc-phosphotransferase also comprises a gamma subunit, which is encoded by: (1) a sequence comprising 1219 bp; or (2) a sequence that hybridizes under stringent conditions to the complement of (1). The deoxymannojirimycin comprises 0.1-5 mM. The kifunensine comprises 0.1-10 microgram/ml. The phosphodiester alpha-GlcNAcase comprises a sequence having 515 amino acids. It is encoded by: (1) a sequence comprising 2183 bp; or (2) a sequence that hybridizes under stringent conditions to the complement of (1). Treating a patient suffering from a **lysosomal** storage disease comprises: (1) culturing a **lectin resistant** mammalian cell in the presence of deoxymannojirimycin and kifunensine to inhibit glycosylation of the glycoprotein; (2) collecting the **high mannose** glycoprotein; (3) collecting the **lysosomal** hydrolase from the **lectin resistant** cells; (4) contacting the collected **lysosomal** hydrolase with a GlcNAc-phosphotransferase; and (5) contacting the **lysosomal** hydrolase with a phosphodiester alpha GlcNAcase after the contacting with a GlcNAc-phosphotransferase. The method further comprises: (1) transferring a N-acetylglucosamine-1-phosphate from UDP-GlcNAc to the glycoprotein; (2) purifying the glycoprotein comprising N-acetylglucosamine-1-phosphate; and (3) removing a N-acetylglucosamine from the glycoprotein.

ACTIVITY - Gastrointestinal. No biological data given.

MECHANISM OF ACTION - Gene therapy.

USE - The method is useful for producing a **high mannose** glycoprotein (claimed) in a complex carbohydrate deficient cell for treating **lysosomal** storage disease. (46 pages)

L73 ANSWER 3 OF 56 HCAPLUS COPYRIGHT 2004 ACS on STN

AB The novel antiviral protein cyanovirin-N (CV-N) was initially discovered based on its potent activity against the human immunodeficiency virus (HIV). Subsequent studies identified the HIV envelope glycoproteins gp120 and gp41 as mol. targets of CV-N. More recently, mechanistic studies have shown that certain **high-mannose** oligosaccharides (oligomannose-8 and oligomannose-9) found on the HIV envelope glycoproteins comprise the specific sites to which CV-N binds. Such selective, carbohydrate-dependent interactions may account, at least in part, for the unusual and unexpected spectrum of antiviral activity of CV-N described herein. We screened CV-N against a broad range of respiratory and enteric viruses, as well as flaviviruses and herpesviruses. CV-N was inactive against rhinoviruses, human parainfluenza virus, respiratory syncytial virus, and enteric viruses but was moderately active against some herpesvirus and hepatitis virus (bovine viral diarrhea virus) strains (50% effective concentration [EC50] = .apprx.1 μg/mL) while inactive against others. Remarkably, however, CV-N and related homologs showed highly potent antiviral activity against almost all strains of influenza A and B virus, including clin. isolates and a neuraminidase inhibitor-resistant strain (EC50 = 0.004 to 0.04 μg/mL). When influenza virus particles were pretreated with CV-N, viral titers were lowered significantly (>1,000-fold). Further studies identified influenza virus hemagglutinin as a target for CV-N, showed that antiviral

activity and hemagglutinin binding were correlated, and indicated that CV-N's interactions with hemagglutinin involved oligosaccharides. These results further reveal new potential avenues for antiviral therapeutics and prophylaxis targeting specific oligosaccharide-comprised sites on certain enveloped viruses, including HIV, influenza virus, and possibly others. hemagglutinin.

L73 ANSWER 4 OF 56 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AB Proteus mirabilis is a common cause of urinary tract infection in individuals with structural abnormalities or long-term catheterization. MR/P fimbria, a **mannose-resistant hemagglutinin**, contributes to colonization of urinary tract by P. mirabilis in a murine model. The expression of MR/P fimbriae is phase-variable. The promoter for the mrp operon, which contains all the genes required for MR/P fimbriae assembly, resides on a 252-bp invertible element flanked by a pair of 21-bp inverted repeats. Previous studies have shown that mrpI, which is transcribed divergently from the mrp operon, encodes a recombinase capable of switching the invertible element either from ON (an orientation that allows for the transcription of the mrp operon) to OFF (an orientation that prohibits the transcription of the mrp operon) or from OFF to ON. In this study, we constructed isogenic mrpI null mutants from a clinical isolate of P. mirabilis, HI4320. A PCR-based assay using DNA isolated from bacterial cultures grown in vitro showed that the invertible element in the isogenic mrpI null mutants is either locked on or locked off. Assays from urine, bladder, and kidneys of experimentally infected mice indicated that the invertible element of the locked-on mutant remained in the ON orientation whereas the invertible element of the locked-off mutant remained in the OFF orientation during the course of infection. These results demonstrated that MrpI is the sole recombinase that regulates the phase-variable expression of the MR/P fimbriae.

L73 ANSWER 5 OF 56 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AB The papA gene in uropathogenic Escherichia coli strains was characterized by PCR-restriction fragment length polymorphism (RFLP) and enterobacterial repetitive intergenic consensus (ERIC)-PCR. One hundred four E. coli strains from patients with urinary tract infections and 32 strains from healthy persons were examined. Thirty seven (27.2%) strains (33 from patients, 4 from healthy persons) in the 136 E. coli strains were positive in **mannose resistant hemagglutination** (MRHA) test. The adherence of MRHA positive strains to HEp-2 cells was greater than those of MRHA negative isolates ($p<0.001$). PapA-PCR were positive in 25% (26/104) of the strains from patients, and 3.1% (1/32) of the strains from healthy persons. Among 27 papA-positive isolates, subtypes were identified by RFLP as 8 (29.6%) F72, 3 (11.1%) F9, 4 (14.8%) F12, and 4 (14.8%) F13. Six groups with novel RFLP patterns were detected, also. The subtypes of P-fimbriae was highly similar to each other by ERIC-PCR.

L73 ANSWER 6 OF 56 MEDLINE on STN DUPLICATE 3
AB Fimbriation, hemagglutination and adherence properties were studied in two strains of S. marcescens (ATCC 43820 and 43821) isolated from the urine of two hospitalized patients in two different hospitals. Studies were performed using electron microscopy (EM), fimbrial purification, **recombinant** DNA and hemagglutination techniques, hydrophobicity and tests of adherence to uroepithelial cells, catheters and glass. In EM, fimbriae of these two strains showed an inner channel and were 11 nm. thick and 0.76-1.08 microns long. Original strains and the clone GH42-pSF192 (**recombinant** DNA prepared using E. coli GH42 as recipient and the cosmid SuperCos 1 as a vector) versus negative control (E. coli GH42-SuperCos 1) showed **mannose-resistant hemagglutination** of tanned erythrocytes and yeast, high hydrophobicity (55.4 and 49.6% at 37C versus 22.8%) and high adherence to borosilicate glass (313,000 and 168,000 CFU/cm.2 versus 17,000 CFU/cm.2), catheters (4.7 x 10(6) and 1.0 x 10(6) CFU/cm.2 versus 3.9 x 10(4)

CFU/cm.2) and uroepithelial cells (adherence indexes of 3.82 and 3.29 versus 1.25). The properties of the fimbriae studied were different from those previously described in the genus Serratia, and they were designated as MR/T.

L73 ANSWER 7 OF 56 Elsevier BIOBASE COPYRIGHT 2004 Elsevier Science B.V. on STN

AB Fifty strains of Escherichia coli isolated from coliseptcemic chickens in Londrina, Brazil, were examined for presence of gene sequences for pil and pap, hemagglutination, and adherence to chicken tracheal cells. Forty-one strains were pil.sup.+ and 22 of these showed **mannose** sensitive (MS) hemagglutination (MSHA) of guinea-pig erythrocytes, indicating that they possessed only type I pill. Seven strains were pap.sup.+ and 6 of these caused **mannose resistant** (MR) **hemagglutination** (MRHA) of human erythrocytes. Twenty-four strains (17 of which caused MSHA) showed MS-adherence to chicken tracheal cells and the remaining 26 showed MR-adherence. The former typically adhered to the mucus layer whereas the latter usually adhered to the mucosal epithelium. It is concluded that MS adherence to chicken tracheal cells is correlated with expression of type 1 fimbriae and that MR-adherence to chicken tracheal cells cannot always be attributed to P pili.

L73 ANSWER 8 OF 56 MEDLINE on STN DUPLICATE 4

AB N-Acetylglucosaminyltransferase V (GlcNAc-T V) is a glycosyltransferase which transfers N-acetylglucosamine in beta(1,6) linkage to the alpha(1,6)-linked **mannose** residue of Asn-linked oligosaccharides. This enzyme is characterized by several unusual properties: GlcNAc-T V is the largest lumenal Golgi glycosyltransferase described thus far, and its multiple mRNA transcripts range from 4.5 to about 9.5 kb; GlcNAc-T V mRNA and activity are regulated by the src tyrosine kinase signalling pathway; in brain tissue, large levels of GlcNAc-T V mRNA are present, but only relatively low levels of catalytic activity can be detected; a **lectin-resistant** cell line, Lec4A, expresses active GlcNAc-T V which is mislocalized intracellularly. In addition, the cell surface oligosaccharide products of this enzyme have been hypothesized to regulate intercellular adhesion. In order to devise specific inhibitors of this enzyme it is necessary to understand its physical structure and how structural changes can influence its activity and localization. We have expressed milligram amounts of a soluble form of **recombinant** rat GlcNAc-T V, purified it from CHO cell-conditioned media, and used it to prepare specific antisera. This antisera binds selectively to GlcNAc-T V and has been used to visualize B-16 mouse melanoma cell GlcNAc-T V on immunoblots after SDS-PAGE. When the antisera was used in immunofluorescence microscopy experiments on permeabilized B-16 and baby hamster kidney cells, intense, specific staining was observed in intracellular structures which appear to correspond to the Golgi apparatus.

L73 ANSWER 9 OF 56 MEDLINE on STN DUPLICATE 5

AB The gene encoding a **mannose-resistant** **hemagglutinating** protein was cloned from Escherichia coli O9:H10:K99. The hemagglutinin is different from two other **mannose** **-resistant hemagglutinins** in this strain, K99 and F41. The agglutinin, named heat-resistant agglutinin 1 (HRA1) since heating to 70 degrees C does not destroy its aggregative properties, strongly agglutinates human, pig, and dog erythrocytes, shows little or no affinity towards cow and chicken erythrocytes, but agglutinates human colon adenocarcinoma 201 (COLO 201) cells. The hra1 gene present on the **recombinant** plasmid pETE1 was localized by subcloning, and its nucleotide sequence was determined. The gene consists of a 792-bp open reading frame coding for a putative protein of 29 kDa with a predicted N-terminal secretory signal sequence. HRA1 shares no significant identity with data base protein sequences. HRA1 is strongly associated with the

bacterial membrane, resisting sonication and isolation attempts based upon standard adhesin purification techniques. N-terminal sequencing of a unique 25-kDa band present in polyacrylamide gels of outer membrane preparations of bacteria harboring pETE1 correlated with the predicted N-terminal amino acid sequence of HRA1 after cleavage of the signal peptide. A viscometric agglutination assay sensitive to the strength of bacterial adhesion shows that the agglutination mediated by bacteria expressing HRA1 is weaker than that of bacteria bearing the F41 adhesin, probably because of the high-molecular-weight, multivalent nature of the latter adhesin. Our observations suggest that HRA1 is a monomeric outer membrane agglutinin.

L73 ANSWER 10 OF 56 MEDLINE on STN DUPLICATE 6

AB An endogenous Madin-Darby canine kidney (MDCK) **lysosomal** membrane glycoprotein that exhibits a basolateral targeting pathway to the **lysosome** is shown here to exhibit significant N-terminal amino acid sequence identity to **lysosomal** associated membrane proteins (LAMP-2) of other species. During establishment of the MDCK monolayer after only 1 d of culture, this canine LAMP-2 has a larger molecular size (110 kDa) than following formation of a confluent monolayer after 3 d of culture (100 kDa) due to the increased presence of N-linked polylactosamine oligosaccharide chains. Neither polylactosamine glycosylation of LAMP-2 in MDCK cells nor truncation of N-linked oligosaccharide chains of LAMP-2 in a **ricin-resistant** MDCK-RCAR cell line influenced the basolateral polarity of its targeting. However, the rate of basolateral delivery of LAMP-2 in MDCK cells plated for 3 d was significantly faster (t1/2 = 28 min) than in 1-d cells (t1/2 = 40 min); in MDCK-RCAR cells the rate of basolateral delivery at both 1 and 3 d of plating was similar (t1/2 = 40 min). The rate differential in MDCK cells occurred after arrival of LAMP-2 to the Golgi apparatus because the rate of acquisition of endoglycosidase H resistance was the same (t1/2 = 25 min) at both days of plating. The rate of transit of LAMP-2 through the Golgi apparatus to the basolateral domain was therefore far more rapid (approximately 4-fold) in 3 d compared with 1-d MDCK cultures. The increased polylactosamine glycosylation of MDCK LAMP-2 at early times of plating during the establishment of a confluent epithelial monolayer may thus be related to its longer residence time in the Golgi apparatus.

L73 ANSWER 11 OF 56 HCAPLUS COPYRIGHT 2004 ACS on STN

AB Ricin is a potent plant toxin that inhibits protein synthesis of eukaryotic cells resulting eventually in cell death. The mechanism of ricin-mediated cytotoxicity on human tumor cell lines was examined in view of the recent findings that bacterial toxins like diphtheria toxin (DTX) and Pseudomonas aeruginosa exotoxin A (PEA) cause programmed cell death or apoptosis in addition to their activity of inhibition of protein synthesis. Ricin is shown to be cytolytic in a short term assay (≤24 h) and cytostatic in a long term assay (≥72 h) as determined by the MTT assay. The cytostatic activity of ricin paralleled its protein synthesis inhibitory activity whereas ricin-mediated cytolytic activity did not correlate with protein synthesis inhibition. Ricin is shown to mediate programmed cell death or apoptosis against several drug sensitive and drug resistant clel lines. DNA fragmentation is initially detected following 4 h of treatment. Although cytotoxicity by ricin is energy dependent, it is not reduced by the addition of a **lysosomotropic** agent NH4Cl. We have recently demonstrated that the bacterial toxins DTX and PEA synergize with tumor necrosis factor-alpha (TNF-α) (J Immunol 147: 2609-2616, 1991 and J Immunol 149:2089-2094, 1992). However, unlike DTX or PEA, ricin did not synergize with TNF-α, suggesting that ricin and DTX/PEA may mediate their cytolytic activity by different mechanisms. These findings demonstrate that ricin can mediate both protein synthesis inhibition and programmed cell death or apoptosis against a variety of drug sensitive and resistant human tumor cell lines. The significance of these findings in cancer therapy is discussed.

L73 ANSWER 12 OF 56 HCAPLUS COPYRIGHT 2004 ACS on STN
AB **Recombinant** K99 and F41 fimbriae antigens expressed by Escherichia coli RRI (pMG611) strain were purified by heat treatment and then by Sephadex G-100 filtration. Their mol. weight was determined by SDS-PAGE.
They correspond to fimbrial antigens from wild strains. The **mannose-resistant hemagglutinating** activity properties of **recombinant** antigens were similar to those of wild strains. Immunol. characteristics of the **recombinant** fimbriae were demonstrated by double immunodiffusion test and Western blot assay. They were also similar to those of wild type fimbriae. The high titer of antibody to both K99 and F41 in rabbit showed that they were strongly immunogenic.

L73 ANSWER 13 OF 56 MEDLINE on STN DUPLICATE 7
AB Nontypeable Haemophilus influenzae M37 adheres to human buccal epithelial cells and exhibits **mannose-resistant hemagglutination** of human erythrocytes. An isogenic variant of this strain which was deficient in hemagglutination was isolated. A protein with an apparent molecular weight of 22,000 was present in the sodium dodecyl sulfate-polyacrylamide gel profile of sarcosyl-insoluble proteins from the hemagglutination-proficient strain but was absent from the profile of the isogenic hemagglutination-deficient variant. A monoclonal antibody which reacts with the hemagglutination-proficient isolate but not with the hemagglutination-deficient isolate has been characterized. This monoclonal antibody was employed in an affinity column for purification of the protein as well as to screen a genomic library for **recombinant** clones expressing the gene. Several clones which contained overlapping genomic fragments were identified by reaction with the monoclonal antibody. The gene for the 22-kDa protein was subcloned and sequenced. The gene for the type b pilin from H. influenzae type b strain MinnA was also cloned and sequenced. The DNA sequence of the strain MinnA gene was identical to that reported previously for two other type b strains. The DNA sequence of the strain M37 gene is 77% identical to that of the type b pilin gene, and the derived amino acid sequence is 68% identical to that of the type b pilin.

L73 ANSWER 14 OF 56 SCISEARCH COPYRIGHT 2004 THOMSON ISI on STN
AB Digalactoside-binding (Gal-Gal) pili and alpha-hemolysin of Escherichia coli have been implicated as important virulence determinants in the pathogenesis of human ascending, nonobstructive pyelonephritis. The pathogenic significance of these determinants was evaluated in vitro and in the BALB/c mouse pyelonephritis model by employing wild-type, avirulent laboratory, and genetically defined cosmids, transformants, and **recombinant** strains. In vitro data suggest that the cytolytic activity of hemolysin is significantly ($P < 0.05$) enhanced among digalactoside-binding strains which agglutinate erythrocytes. The basis of increased hemolysis is related presumably to more efficient delivery of the toxin to target lipid substrate in the host plasma membrane. Intravesicular administration of bacteria that express both digalactoside binding and hemolysin generally resulted in greater mortality and renal parenchymal injury in mice than strains that expressed none or only one of these determinants. Analyses convincingly demonstrate that digalactoside-binding pili are corelated with upper urinary tract colonization and that hemolysin is correlated with septicemia and renal parenchymal damage. These determinants collectively constitute the minimal virulence factors to produce disease in this model. Their efficacy as vaccines for the prevention of pyelonephritis was also assessed. A purified Gal-Gal pilus vaccine prevented ($P < 0.05$) subsequent colonization by a challenge wild-type strain that exhibited homologous pili. The hemolysin vaccine did not abrogate subsequent bacterial renal colonization on challenge, but it did protect ($P < 0.05$) mice which survived challenge from subsequent renal injury compared with those in the saline control group. The combination of these determinants

was also protective. The combination of Gal-Gal pili and hemolysin in a vaccine preparation represents a potentially worthwhile strategy for human immunoprophylaxis against pyelonephritis by interdicting several steps in the pathogenesis of a bacterial mucosal infection.

L73 ANSWER 15 OF 56 SCISEARCH COPYRIGHT 2004 THOMSON ISI on STN

L73 ANSWER 16 OF 56 MEDLINE on STN

AB The Dr hemagglutinin of uropathogenic Escherichia coli mediates adherence to the upper urinary tract. E. coli strains which express this adhesin bind to the Dr blood group antigen and mediate **mannose-resistant hemagglutination** (MRHA). Chloramphenicol inhibits MRHA produced by the Dr hemagglutinin and may act as an analog for the tissue receptor at the adhesin-binding site. The nucleotide sequence of the Dr hemagglutinin fimbrial subunit was determined and found to have significant homology with that of F1845, a fimbrial adhesin associated with diarrhea, and with the afimbrial adhesin AFA-I of uropathogenic E. coli. Chimeric adhesin determinants consisting of the Dr structural subunit and F1845 accessory genes or of the F1845 structural subunit and Dr accessory genes were constructed. The Dr and F1845 determinants were shown to have a close structural relationship, with functional differences concentrated in the fimbrial subunit. Oligonucleotide-directed site-specific mutagenesis was used to facilitate construction of a hybrid adhesin subunit gene containing the amino terminus of F1845 fused to the carboxy terminus of the Dr structural gene. The resulting construct confers chloramphenicol-**resistant hemagglutination** when introduced into an E. coli strain expressing the cloned Dr hemagglutinin. The chloramphenicol sensitivity or resistant phenotype of MRHA produced by this family of adhesins is determined solely by the fimbrial subunit gene. Domains responsible for the chloramphenicol sensitivity of Dr-mediated MRHA reside within the amino-terminal portion of the fimbrial subunit.

L73 ANSWER 17 OF 56 SCISEARCH COPYRIGHT 2004 THOMSON ISI on STN

AB Brefeldin A (BFA), a drug that induces redistribution of Golgi-apparatus proteins into the endoplasmic reticulum, was used to determine the role of subcellular compartmentalization in the processing of asparagine-linked oligosaccharides. Baby-hamster kidney cells were pulse-labelled with [H-3]mannose for 30-60 min and chased for up to several hours in the presence or in the absence of BFA or labelled continuously for several hours with and without the drug. Cellular glycoproteins were digested to glycopeptides with Pronase and either fractionated into glycan classes by lectin affinity chromatography or digested further by endoglycosidase H and endoglycosidase D. Released oligosaccharides obtained in the latter procedure were then separated from each other and from endoglycosidase-resistant glycopeptides by paper chromatography. The results show that BFA induces a very fast processing of protein-linked Glc3Man9GlcNAc2 oligosaccharide down to Man5GlcNAc2 and conversion into complex-type and hybrid-type glycans. The major difference between untreated and BFA-treated cells is a large increase in bi-antennary and hybrid-type glycans in the latter cells. These results indicate that galactosylation of a mono-antennary GlcNAcMan5GlcNAc2 hybrid blocks subsequent action by mannosidase II and N-acetylglucosaminyl transferase II, producing galactosylated hybrid-type glycans. Similarly, galactosylation of the product of N-acetylglucosaminyltransferases I and II, i.e. a Man3GlcNAc2 core substituted with GlcNAc-beta-1 --> 2 on both alpha-1 --> 3- and alpha-1 --> 6-linked mannose residues, blocks branching N-acetylglucosaminyltransferases IV and V, thereby causing an increase in bi-antennary glycans and a decrease in tri- and tetra-antennary glycans.

L73 ANSWER 18 OF 56 HCAPLUS COPYRIGHT 2004 ACS on STN

AB A review with 125 refs. Enterotoxigenic E. coli (ETEC) strains may synthesize both thermolabile (LT-I and LT-II) and thermostable (STa and STb) enterotoxins. In addition to the synthesis of enterotoxins, the ability

of ETEC strains to cause diarrhea is also conditioned by the possession of colonization factors which enable bacteria to adhere to and colonize the luminal surface of the small bowel. Colonization factors in ETEC strains were located in rigid fimbriae and flexible fibrils constituted by protein subunits ranging in size from 14,500 d to 31,000 d and usually responsible for **mannose-resistant hemagglutination** with determined erythrocyte species. Both enterotoxins and colonization factors are controlled by plasmids. There exist plasmids which may code sep. enteroxins and colonization factors, and besides there also exist **recombinant** plasmids in coding both of these two virulence factors. Human ETEC strains may synthesize LT-I and/or STa enterotoxins, they may possess the colonization factors named CFA/I, CFA/II, CFA/III, or CFA/IV, and they belong mainly to serogroups O6, O8, O15, O20, O25, O27, O63, O77, O78, O114, O115, O126, O128, O139, O148, O153, O159, and O167. ETEC strains from porcine origin synthesize LT-I, STa, and/or STb, they possess the colonization factors K88, P987, K99, or F41, and they usually belong to serogroups O8, O9, O20, O45, O64, O101, O115, O138, O141, O147, O149, and O157. Bovine and ovine ETEC strains are usually STa producers harboring on the bacterial surface K99 or F41 colonization factors, and they belong to serogroups O8, O9, and O101. Nevertheless, some particular bovine ETEC strains synthesizing LT-II have been described. Thus, a high specificity level between ETEC strains causing diarrhea in humans and domestic animals can be observed This is mainly due to the specific recognition between bacterial colonization factors and the epithelial receptors during host-parasite interaction.

L73 ANSWER 19 OF 56 MEDLINE on STN DUPLICATE 8

AB Normal bovine and mouse sera contain a component, termed beta inhibitor, that inhibits the infectivity and hemagglutinating activity of influenza A viruses of the H1 and H3 subtypes. To investigate the nature of the interaction of beta inhibitors with influenza A viruses we isolated a mutant of the virus Mem71H-BelN (H3N1) that could grow in the presence of bovine serum. The mutant virus was **resistant** to **hemagglutination** inhibition by mouse serum as well as by bovine serum and had undergone changes in the receptor-binding and the antigenic properties of its hemagglutinin (HA) molecule. Sequence analysis of the HA genes of parent and mutant viruses revealed a single nucleotide change in the mutant, resulting in the substitution Thr----Asn at residue 167 of the HA1 chain of HA. This change leads to loss of the potential glycosylation site Asn-165-Val-166-Thr-167 at the tip of the HA spike, which in viruses of the H3 subtype is known to bear a **high-mannose** (type II) carbohydrate side chain N-linked to Asn-165. The association of beta inhibitor resistance with loss of this carbohydrate side chain suggested that beta inhibitors may be lectins. In support of this hypothesis, treatment of the beta inhibitor-sensitive parent virus Mem71H-BelN with periodate converted it to the resistant state. Furthermore, the inhibitory activity of both bovine and mouse sera for the parental virus was abrogated by D-mannose. We conclude that the beta inhibitors in bovine and mouse sera are mannose-binding lectins that inhibit hemagglutination and neutralize virus infectivity by binding to carbohydrate at the tip of the HA spike, blocking access of cell-surface receptors to the receptor-binding site on HA.

L73 ANSWER 20 OF 56 MEDLINE on STN

AB Acute pyelonephritis, a complication of Escherichia coli bacteriuria, must represent a bacterial invasion through the kidney epithelium. To study this process, we overlaid bacterial suspensions onto monolayers of cultured human kidney proximal tubular epithelial cells and measured cytotoxicity by release of lactate dehydrogenase (LDH). Thirty-four isolates cultured from patients with acute pyelonephritis were screened for the ability to cause pyelonephritis in CBA mice by transurethral challenge. The eight most virulent strains (greater than or equal to 70% of mice challenged developed greater than or equal to 10(3) CFU/g of kidney after 48 h) were selected for study. Each strain displayed

mannose-resistant hemagglutination of human O erythrocytes; three strains were phenotypically and genotypically hemolytic. Pyelonephritogenic strains were significantly more cytotoxic (30.1 +/- 9.5% LDH release after 18 h) than eight fecal control strains (13.5 +/- 11.5% LDH release; P = 0.0068). We selected the most cytotoxic strain, CFT073, for further study. Sterile filtrate from this hemolytic strain was significantly more cytotoxic than was the filtrate of the fecal control strain, FN414. Transposon mutagenesis of CFT073 with TnphoA abolished hemolytic activity and cytotoxicity by both whole cells and sterile filtrate. Southern blot analysis revealed that the Tnphoa insertion mapped to the E. coli chromosomal hly determinant within a 12-kilobase SalI restriction fragment. Transformation of a nonhemolytic strain, CPZ005 with plasmid pSF4000, which carries a cloned hemolysin determinant, resulted in highly elevated cytotoxicity. Light micrographs of proximal tubular epithelial cell cultures demonstrated cell damage by pyelonephritogenic strains that was not induced by a fecal strain or the hemolysin-deficient mutant. Results indicate that pyelonephritogenic E. coli strains are more frequently cytotoxic for a putative target, that is, human renal tubular epithelium, than are fecal isolates. Hemolysin, in some strains, is apparently responsible for this cytotoxicity.

L73 ANSWER 21 OF 56 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
DUPLICATE 9
AB Epidermal growth factor (EGF) was linked to the toxic A chain of ricin toxin (RTA) to produce an EGF-receptor-specific cytotoxic agent, EGF-RTA. Three EGF-RTA-resistant mutants of the human HeLa cell line were selected. These mutant cell lines are 10-fold to more than 100-fold more resistant to EGF-RTA when compared to HeLa cells. The EGF-RTA-resistant mutants have at least as many EGF receptors as parent cells; the basis for the EGF-RTA-resistant phenotype must be distal to EGF binding. The EGF-RTA-resistant cells are not cross-**resistant** to **ricin** or to diphtheria toxin; their mutant phenotype appears to be EGF specific. The EGF-RTA-resistant mutants are able to internalize and degrade EGF. However, the mutants have altered EGF receptor down-regulation and phorbol 12-tetradecanoate 13-acetate modulation properties. EGF-RTA/ammonium chloride and EGF-RTA/adenovirus co-treatment data suggest that the mutant defect(s) which confers EGF-RTA resistance is either in the endosome or at a step(s) in the intracellular EGF processing pathway between the endosome and the **lysosome.**

L73 ANSWER 22 OF 56 MEDLINE on STN DUPLICATE 10
AB The genetic organization of the polypeptides required for the biosynthesis of the F41 adhesin of enterotoxigenic Escherichia coli strains was investigated. Maxicell analysis demonstrated that a **recombinant** plasmid which mediated **mannose-resistant hemagglutination** and F41 antigen production encoded four polypeptides of 29, 30, 32, and 86 kilodaltons. The 29-kilodalton protein was identified as the F41 antigen, and the nucleotide sequence of the gene was determined. Extensive homology was observed between the region encoding the putative signal sequences of the F41 and K88 antigens and in the region immediately upstream of the antigen genes. The nucleotide sequence homology between F41 and K88 determinants was further investigated by Southern blot hybridization. A K88 probe hybridized at high stringency to all fragments shown to be essential for F41 production except for fragments internal to the F41 antigen gene.

L73 ANSWER 23 OF 56 MEDLINE on STN DUPLICATE 11
AB The afimbrial adhesin (AFA-I) from a pyelonephritic Escherichia coli isolate (KS52) is a **mannose**-resistant, P-independent, X-binding adhesin, expressed by the afa-1 operon. It is distinct from the E. coli X-binding adhesins with M and S specificity. A total of 138 E. coli isolates belonging to various serotypes, mostly from urinary tract infections, were screened for the presence of DNA sequences related to the afa operon and for the expression of an X-adhesin able to mediate

mannose-resistant hemagglutination (MRHA) and adhesion to uroepithelial cells. Fifteen strains were shown to harbor DNA sequences related to the AFA-I-encoding operon, and 13 of them expressed an X-adhesin. Using as probes different DNA segments of the AFA-I-encoding operon in Southern experiments, we demonstrated that only three of these clinical isolates contained genetic determinants closely related to those identified in the original afa prototype strain (KS52): presence of the afaA, afaB, afaC, afaD, and afaE genes associated with the expression of a 16,000-dalton hemagglutinin-adhesin which strongly cross-reacted with AFA-I-specific antibodies. The other E. coli isolates harbored DNA sequences homologous to the afaA, afaB, afaC, and afaD genes, but lacked the sequence corresponding to the adhesin-producing gene afaE; Western blots allowed the detection of polypeptides (15,000, 15,500, or 16,000 daltons) in these strains which cross-reacted with variable intensity with antibodies raised against the denatured AFA-I protein, but did not cross-react with native AFA-I-specific antibodies. Following DNA cloning experiments from chromosomal DNA of two of those strains (A22 and A30), we demonstrated that although the AFA-related operon in A22 and A30 strains lacked the AFA-I adhesin-encoding gene, they synthesized a functional X-adhesin. Thus, strains A22 and A30 encode adhesins designated AFA-II and AFA-III, which were cloned on **recombinant** plasmids pILL72 and pILL61, respectively. Southern hybridization experiments and Western blot analyses of the 15 AFA-related strains demonstrate the heterogeneity of the genetic sequences encoding the structural adhesin and suggest the bases for the serological diversity of the AFA adhesins.

L73 ANSWER 24 OF 56 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AB In this study, the K99 plasmid DNAs from wild strains of enterotoxigenic E. coli from bovine origin were investigated for cloning into E. coli K-12 (HB 101) by using the pBR 322 cloning vector. The results obtained were abstracted as follows: Of 50 strains of E. coli isolated from calves with diarrhea, 31 strains (62%) were proved to possess K99 pilus antigen by serological test. All 9 strains possessing K99 pilus antigens were recognized to produce heat-stable enterotoxins (ST) because the ratios of gut weight to remaining body weight were higher than 0.083. **Mannose-resistant hemagglutinating** activity (MRHA) by using horse red blood cells of 9 strains of E. coli were ranged from 1:8 to 1:64. The K99 plasmid DNA purified from strain (80-56) of E. coli was directly transformed into E. coli K-12 (HB101). The plasmids of the transformants harbored about 85 kilobase (Kb) fragment that encoded for K99 pilus antigens. It was proved to possess DNA fragment which is able to synthesize the K99 pilus antigens by MRHA test using horse red blood cells. The 85 Kb plasmid was cleaved 6 fragments (28, 21, 16.8, 8.5, 6.7 and 4 Kb) when digested with Hind III. Among fragments, 16.8 Kb plasmids were cloned at Hind III site into E. coli K-12 (HB 101) by using the pBR 322 cloning vector for K99 **recombinant** plasmid. One of these clones contained 21.2 Kb **recombinant** plasmid designated pYYD 86 and MRHA of its was 1:128.

L73 ANSWER 25 OF 56 MEDLINE on STN DUPLICATE 12
AB The uropathogenic strain Escherichia coli IH11128 (O75:K5:H-) exhibits a **mannose**-resistant O75X adhesin. The genes encoding the O75X adhesin were cloned from a clinical strain and transferred to E. coli K-12 derivatives. The **recombinant** plasmids were found to express a 15-kilodalton fimbrial subunit protein, a fimbrialike extracellular structure, and **mannose-resistant hemagglutination.** An indirect immunofluorescence assay was used to study attachment of the clone and purified adhesin to frozen sections of human kidney. The clone bound selectively to the interstitial areas and notably to Bowman's capsule. The purified adhesin bound to the basement membrane of the tubules and to Bowman's capsule.

L73 ANSWER 26 OF 56 MEDLINE on STN DUPLICATE 13

AB Baby hamster kidney (BHK) cell extracts catalyze the conversion of [3H]mannose-labelled (Man)5GlcNAc and (Man)6GlcNAc oligosaccharides to a (Man)3GlcNAc species that retains affinity for concanavalin-A-Sepharose and appears to be Man alpha 1----3[Man alpha 1----6]Man beta 1----4GlcNAc. The properties of the (Man)5GlcNAc-hydrolase activity differ from **lysosomal** alpha-mannosidases as well as previously described processing mannosidases acting on oligosaccharide intermediates of N-glycan assembly. Mosquito cell extracts catalyze hydrolysis of (Man)6GlcNAc but lack the (Man)5GlcNAc hydrolase activity detected in BHK cell extracts. Glycopeptide analysis has been carried out on a **ricin-resistant** BHK mutant RicR14 that lacks N-acetylglucosaminyl transferase I and fails to convert oligomannosidic N-glycans to complex-type chains, and mosquito cells that constitutively lack N-acetylglucosaminyl transferase I. In both cell lines, the cellular glycoproteins contain (Man)5GlcNAc oligosaccharide as the major stable component equivalent to a 15-20-fold increase compared with normal BHK cells. Although containing very high amounts of asparagine-linked (Man)5(GlcNAc)2, RicR14 cells exhibit (Man)5GlcNAc hydrolase activity at levels similar to wild-type BHK cells. This result, together with previous work [Foddy, L., Feeney, J. & Hughes, R.C. (1986) Biochem. J. 233, 697-706] showing the complete inhibition of conversion of oligomannosidic intermediates to complex-type N-glycans in BHK cells treated with swainsonine, an inhibitor of mannosidase II but not the (Man)5GlcNAc hydrolase activity, argues against a major role for the (Man)5GlcNAc hydrolase activity in N-glycan assembly and suggesting other functions for the mannosidase activity in BHK cells.

L73 ANSWER 27 OF 56 MEDLINE on STN DUPLICATE 14

AB We investigated the role of bacterial mannose-resistant fimbriation of S fimbriae (Fim), mannose-**resistant hemagglutination** (S-Mrh), and hemolysin (Hly) production by an Escherichia coli parent and genetically cloned strains as regards their effect on histamine release from rat mast cells and generation of the chemiluminescence response, leukotriene, and enzyme release from human polymorphonuclear granulocytes. These mediators are involved in the induction of inflammatory disease processes and lead, e.g., to the enhancement of vascular permeability, chemotaxis, aggregation of granulocytes (leukotriene B4), **lysosomal** enzyme release, and smooth-muscle contraction (leukotrienes C4, D4, and E4). The content of azurophilic and specific granules in polymorphonuclear granulocytes consists of highly reactive enzymes which amplify inflammatory reactions. Washed bacteria (E. coli 764 Hly+/-, E. coli 21085 Hly+/- Fim+/- Mrh+/-), as well as their culture supernatants, were analyzed at various times during their growth cycle. No differences exist between parent and cloned or mutant strains with respect to their outer membrane proteins and lipopolysaccharide pattern. Washed bacteria [E. coli 764 and 21085(pANN202-312)] which produced hemolysin, unlike Hly- strains, induced high levels of histamine release from rat mast cells and led to a significant chemiluminescence response and enzyme and leukotriene release from human polymorphonuclear granulocytes. Bacterial culture supernatants from Hly+ and secreting strains showed similar results with the exception of E. coli 21085(pANN202-312), which is a hemolysin-producing but not a secretory strain. Our data suggest a potent role for hemolysin as a stimulus for noncytotoxic mediator release from various cells. Furthermore, we showed that the presence of Fim and S Mrh potentiates mediator release. The simultaneous presence of Mrh and Fim [E. coli 535/21(pANN801-4)] increased mediator release compared with Mrh+ Fim- strains [E. coli 536/21(pANN801-1)]. E. coli 536/21 (Msh- Mrh- Fim- Hly-) did not induce mediator release.

L73 ANSWER 28 OF 56 MEDLINE on STN

AB The genetic determinant for production of the adhesive antigen F41 was isolated from a porcine enterotoxigenic Escherichia coli strain by cosmid cloning. The cloned DNA included sequences homologous to those of

hybridization probes prepared from the K88 adhesive antigen operon. Transposon insertions which inactivated F41 production mapped to the same region of DNA showing homology with the K88 genes, demonstrating the genetic relatedness of F41 and K88. Hybridization of a K88 gene probe to plasmid and total DNA from the porcine E. coli isolate from which the F41 gene was cloned indicated that F41 is chromosomally encoded by this strain. This observation was extended to other F41-producing animal isolates. A large number of animal E. coli isolates were examined with K88, F41, and K99 gene probes and for **mannose-resistant hemagglutination** of human group O erythrocytes and K88 and F41 antigen production. All K88 and F41 antigen producers possessed genetic homology with the K88 and F41 gene probes. Most, but not all, F41-producing strains possessed homology to the K99 gene probe, reflecting the previously observed association of F41 and K99 antigen production. In the strains examined, homology with the K99 gene probe was plasmid associated, whereas homology with the F41 gene probe was chromosomal. The K88 antigen-producing strains showed no homology with the K99 probe. A number of strains possessed homology with the K88 and F41 gene probes and were **mannose-resistant hemagglutination** positive, but did not produce K88 or F41 antigens. This suggests that there are adhesins among animal isolates of E. coli which are genetically related to but antigenically distinct from K88 and F41.

L73 ANSWER 29 OF 56 MEDLINE on STN DUPLICATE 15

AB Escherichia coli 536 (O6:K15:H31), which was isolated from a case of urinary tract infection, determines high nephropathogenicity in a rat pyelonephritis system as measured by renal bacterial counts 7 days after infection. The loss of S fimbrial adhesin formation (Sfa-) (**mannose-resistant hemagglutination** [Mrh-] and fimbria production [Fim-]), serum resistance (Sre-), and hemolysin production (Hly-) in the mutant 536-21 led to a dramatic reduction of bacterial counts from almost 10(5) to only 40 cells per g of kidney. The reintroduction of the cloned S fimbrial adhesin determinant (sfa) increases the virulence of the avirulent mutant strain by a factor of 20; almost the same effect was observed after restoration of serum resistance by integration of an sfa+ **recombinant** cosmid into the chromosome. Additional reintroduction of the Hly+ phenotype by transformation of two hly determinants increased the virulence of the strains. Hemolysin production determined increased renal elimination of leukocytes and erythrocytes. Thus all three determinants investigated, S fimbriae, serum resistance, and hemolysin, contribute to the multifactorial phenomenon of E. coli nephropathogenicity.

L73 ANSWER 30 OF 56 MEDLINE on STN DUPLICATE 16

AB Baby-hamster kidney (BHK) cells were grown continuously in long-term monolayer culture in the presence of Swainsonine, an inhibitor of alpha-mannosidase II, a processing enzyme involved in glycoprotein biosynthesis. The asparagine-linked oligosaccharides (N-glycans) were isolated from Pronase-digested cells by gel filtration, ion-exchange chromatography and affinity chromatography on concanavalin A--Sepharose and lentil lectin--Sepharose. The major N-glycans, analysed by 500 MHz 1H-n.m.r. spectroscopy, were identified as hybrid structures containing five mannose residues and neutral **high-mannose** N-glycans. The major hybrid species contained a core-substituted fucose alpha(1----6) residue and a NeuNAc alpha(2----3)Gal beta(1----4)GlcNAc terminal sequence; smaller amounts of non-sialylated and non-fucosylated hybrid structures were also detected. Swainsonine-treated cells also produced neutral oligosaccharides containing a single reducing N-acetylglucosamine residue substituted with polymannose sequences. The glycopeptide composition of Swainsonine-treated BHK cells resembles closely that of the **ricin-resistant** BHK cell mutant, RicR21 [P. A. Gleeson, J. Feeney and R. C. Hughes (1985) Biochemistry 24, 493-503], except the hybrid structures of RicR21 cells contain three, not five, mannose residues. Like RicR21 cells, Swainsonine-treated BHK

cells showed a greatly increased **resistance** to **ricin** cytotoxicity, but not to modeccin, another galactose-binding lectin. These effects were readily reversed on removal of Swainsonine and growth in normal medium.

L73 ANSWER 31 OF 56 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AB The usefulness of **mannose** resistant haemagglutination (MRHA) test to detect adhesive antigens of Escherichia coli causing colibacillosis in piglets was estimated using horse, bovine, sheep, swine and guinea pig erythrocytes. One hundred seventy seven isolates from piglets dead due to enteric colibacillosis, 45 isolates from pigs with oedema disease, 39 isolated from faeces of normal piglets and 2 strains, laboratory **recombinants** (incorporated plasmids determining synthesis of K88 and K99 antigens) were examined. It was found that the MRHA test with the use of guinea pig or swine erythrocytes enables to detect enterotoxic strains of E. coli producing adhensive antigens (ETEC) and causing enteritis in piglets and to distinguish E. coli strains causing oedema disease from those nonpathogenic.

L73 ANSWER 32 OF 56 MEDLINE on STN DUPLICATE 17
AB Fibronectin isolated from the conditioned medium of monolayer cultures of baby hamster kidney (BHK) cells and several **ricin-resistant** (Ric) mutants derived from them express differences in N-glycosylation. The asparagine-linked oligosaccharides of BHK cell-derived fibronectin consist largely of complex chains, whereas hybrid and/or **high-mannose** chains are present in the fibronectins of mutant cell lines. The fibronectins exhibiting different glycosylation patterns are incorporated to similar extents into the cell-layer of human skin fibroblasts. In contrast, mutant cells retain significantly less endogenously produced fibronectin than BHK cells and also incorporate less human cellular fibronectin into a pericellular matrix. In vitro adhesion assays show that mutant cells attach to and spread relatively poorly on fibronectin-or type IV collagen-coated substrata but interact as well as do BHK cells with a laminin substratum. These results indicate that asparagine-linked oligosaccharides of fibronectin are not required for the binding and incorporation of the molecule into cell layers, but, as constituents of other cellular glycoproteins, they do modulate the ability of BHK cells to interact with some matrix components.

L73 ANSWER 33 OF 56 MEDLINE on STN DUPLICATE 18
AB MDW4, a wheat germ agglutinin-resistant mutant of the metastatic murine tumor MDAY D2 has previously been shown to be poorly metastatic when injected intravenously and non-metastatic when injected subcutaneously into syngeneic mice. W4EB8, a Bandeiraea simplicifolia (BSII) lectin-selected subline of MDW4 has previously been shown to be intermediate between that of MDAY-D2 and MDW4 cell for sensitivity to lectin and metastatic phenotype when injected intravenously into mice. The Asn-linked oligosaccharides from MDAY-D2, MDW4 and W4EB8 cells were released enzymatically with peptide N-glycosidase, reduced with tritiated sodium borohydride and fractionated by Concanavalin-A--Sepharose affinity chromatography and high-performance liquid chromatography (HPLC). Structures of the major fractions were determined by a combination of glycosidase digestion and sizing, gas-liquid chromatography/mass spectrometry and by proton nuclear magnetic resonance. Wild-type and mutant cells processed **high-mannose**-type structures to biantennary (GlcNAc)2(Man)3(GlcNAc)2. In MDAY-D2 cells this structure was processed further to sialylated tetra-antennary complex with polylactosamine-containing antennae terminating in either sialic acid or alpha 1-3-linked galactose. MDW4 cells had four or five times more (GlcNAc)2(Man)3(GlcNAc)2 than MDAY-D2 cells and a major component of tri-antennary (GlcNAc)3(Man)3(GlcNAc)2 (i.e. 2,2,6-substituted tri-mannosyl core) that was not found in wild-type cells. The partial revertant, W4EB8 had intermediate levels of mutant

(GlcNAc)3(Man)3(GlcNAc)2 and sialylated complex-type carbohydrates. The results indicate that a shift in expression from incomplete complex type to sialylated tri/tetra-antennary complex-type carbohydrates in tumor cell may enhance the metastatic potential of tumor cells in the experimental metastasis assay. In addition, somatic cell hybridization analysis indicated that the defect in MDW4 cells was identical to that of the Chinese hamster ovary mutant Lec8: a deficiency in UDP-galactose transport into the golgi.

L73 ANSWER 34 OF 56 HCAPLUS COPYRIGHT 2004 ACS on STN
AB It was previously reported (Dall' Olio, F. et al., 1986) that the major species of **high-mannose** chains accumulating in the viral glycoprotein C from **ricin-resistant** cells was Man4GlcNAc2. When the sample was analyzed by using HPLC, it was found that the major species is Man5GlcNAc2.

L73 ANSWER 35 OF 56 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN

L73 ANSWER 36 OF 56 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED. on STN DUPLICATE 19
AB The progeny of Herpes simplex virus type 1 (HSV-1) grown in **ricin -resistant** 14 cells (Ric(R)14) lacking N-acetylglucosaminyltransferase I was released in the extracellular medium at a very low rate. By using a monoclonal antibody immobilized on Sepharose we purified from HSV-1-infected Ric(R)14 cells a viral glycoprotein (gC), which carries both N- and O-linked oligosaccharides. Glycopeptides obtained from [3H]mannose-labeled gC by Pronase digestion were entirely susceptible to endo-β-N-actylglucosaminidase H, and the major oligosaccharide released was Man4GlcNAc. The accumulation of this **high-mannose** species was related to the enzymic defect of the host cells and to the long retention of the viral glycoprotein within the cells. The wextent of O-glycosylation evaluated in [14C]glucosamine-labeled gC from Ric(R)14 cells as compared to that of gC from wild type cells did not appear to be significantly modified.

L73 ANSWER 37 OF 56 MEDLINE on STN DUPLICATE 20
AB The asparagine-linked glycopeptides (N-glycans) of a **ricin-resistant** mutant of baby hamster kidney (BHK) cells, RicR21, have been isolated and fractionated from a Pronase digest of disrupted cells by concanavalin A (Con A)-Sepharose chromatography, ion-exchange chromatography, and lentil lectin chromatography. The structures of all the major N-glycans have been determined by 500-MHz H NMR spectroscopy. RicR21 synthesizes only hybrid and **high-mannose** N-glycans. All the hybrid structures contain only three mannose residues. The major hybrid glycopeptide has the following structure: (Formula: see text). There is also about 15% of the nonfucosylated species present. Only a small amount (less than or equal to 5%) of the asialo hybrid is produced. Branched hybrid N-glycans are also present in RicR21 cells, containing two complex antenna linked beta 1----2 and beta 1----4 to the Man alpha 1----3 arm; about 70% of this species is core fucosylated. Man6GlcNAc2 glycopeptide is the most abundant (about 70%) of the **high-mannose** N-glycans. These studies account for the very poor ricin binding property of this mutant, as the sialic acid residues of the major hybrid N-glycan are exclusively linked alpha 2----3 to galactose and ricin is unable to bind to alpha 2----3-substituted galactosyl residues [Baenziger, J. U., & Fiete, D. (1979) J. Biol. Chemical 254, 9795-9799].

L73 ANSWER 38 OF 56 MEDLINE on STN DUPLICATE 21
AB Recently, several investigators have explored the possibility of targeting ricin to designated cell types in animals by its linkage to specific antibodies. There is evidence, however, that the mannose-containing oligosaccharide chains on ricin are recognised by reticuloendothelial cells in the liver and spleen and so cause the immunotoxins to be removed

rapidly from the blood stream. In the present study we analysed the carbohydrate composition of ricin and examined enzymic methods for removing the carbohydrate. The carbohydrate analysis ricin A-chain revealed the presence of one residue of xylose and one of fucose in addition to mannose and N-acetylglucosamine which had been detected previously. The B-chain contained only mannose and N-acetylglycosamine. Ricin A-chain is heterogeneous containing two components of molecular weight 30 000 and 32 000. Strong evidence was found that the heavier form of the A-chain contains an extra carbohydrate unit which is heterogeneous with respect to concanavalin A binding and sensitivity to endoglycosidase H. The lower molecular weight form of A-chain did not bind concanavalin A and was insusceptible to endoglycosidases. Only one of the two **high mannose** oligosaccharide units on the isolated B-chain could be removed by endoglycosidases H or F, whereas both were removable after denaturation of the polypeptide by SDS. Both the isolated A- and B-chains were sensitive to alpha-mannosidase. Intact **ricin** was **resistant** to endoglycosidase treatment and was only slightly sensitive to alpha-mannosidase. The addition of SDS allowed endoglycosidase H to remove both of the B-chain oligosaccharides from intact ricin and increased the toxin's sensitivity to alpha-mannosidase. In conclusion, extensive enzymic deglycosylation of ricin may only be possible if the A- and B-chains are first separated, treated with enzymes and then recombined to form the toxin.

L73 ANSWER 39 OF 56 HCAPLUS COPYRIGHT 2004 ACS on STN
AB The genes coding for F11 fimbriae from the uropathogenic E. coli C1976 were cloned by a cosmid cloning procedure. Two cosmid clones expressed F11 fimbriae, and these clones possessed an identical DNA fragment of 8.9 kilobases. This fragment was subcloned into pBR322, and the **recombinant** plasmid encoded fimbriae and caused a **mannose -resistant hemagglutination** (MRHA). Polyclonal and monoclonal antibodies were produced against purified cloned F11 fimbriae. Both types of antibodies were used in inhibition tests of MRHA and adherence of bacteria to the uroepithelial cell line T24. After preincubation of bacteria with polyclonal antiserum, the MRHA and the **mannose**-resistant (MR) adherence were totally inhibited. Preincubation of bacteria with monoclonal antibodies did not inhibit MRHA and MR adherence.

L73 ANSWER 40 OF 56 MEDLINE on STN DUPLICATE 22
AB MDW40, a wheat germ agglutinin-resistant (WGAr) mutant of the highly metastatic tumor cell line called MDAY-D2, is restricted to local growth at the subcutaneous site of inoculation. The WGAr tumor cells acquire metastatic ability by fusing spontaneously with a normal host cell followed by chromosome segregation, a process accompanied by reversion of the WGAr phenotype (i.e., WGAs). Since **lectin-resistant** mutant cell lines often have oligosaccharide alterations that may affect membrane function and consequently metastatic capacity, we compared the major Asn-linked glycopeptides in WGAr and WGAs cell lines. [2-3H]mannose-labeled glycopeptides were separated into four fractions on a DEAE-cellulose column and then further fractionated on a concanavalin A-Sepharose column. Glycopeptide structures were determined by: (a) sequential exoglycosidase digestion followed by chromatography on lectin/agarose and Bio-Gel P-4 columns and (b) proton nuclear magnetic resonance analysis. The metastatic WGAs cells had a sialylated poly-N-acetyllactosamine-containing glycopeptide which was absent in the nonmetastatic mutant cell line. Unique to the mutant was a neutral triantennary class of glycopeptide lacking sialic acid and galactose; the WGAr lesion therefore appeared to be a premature truncation of the antennae of the poly-N-acetyllactosamine-containing glycopeptide found in the WGAs cells. **High mannose** glycopeptides containing five to nine mannose residues constituted a major class in both WGAr and WGAs cells. Lysates of both wild-type and mutant cells had similar levels of galactosyltransferase activity capable of adding galactose to the

N-acetylglucosamine-terminated glycopeptide isolated from mutant cells; the basis of the WGAr lesion remains to be determined.

L73 ANSWER 41 OF 56 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
DUPLICATE 23
AB The genes responsible for the formation of the F72 fimbriae of the uropathogenic E. coli strain AD110 (O6:K2:H1:F7) were cloned on the **recombinant** plasmid pPIL 110-35. The F72 fimbriae, like the F71 fimbriae of AD110, are responsible for **mannose resistant hemagglutination** (MRHA). The molecular organization of the genes of pPIL110-35 involved in the expression of MRHA was studied by analysis of transposon γδ and Tn5 insertion mutants. Mutations that cause an MRHA-deficient phenotype were located in discrete groups within an 11.5 kilobase restriction fragment of pPIL110-35, separated by insertion mutations that do not inactivate MRHA. Complementation experiments were also conducted. Restriction fragments of pPIL110-35 subcloned in the vector pBR322 were tested for their ability to complement transposon insertion mutations in the corresponding regions of pPIL110-35. Five complementation groups were distinguished. Five genes (designated A-E) involved in the expression of MRHA can be distinguished by these results. The products of these genes were analyzed in minicells. Gene B apparently codes for a 75 K dalton protein, gene C for a 23 K dalton protein and gene E for a 36 K dalton protein. No product of gene D was observed. Gene A probably codes for the 17 K dalton subunit polypeptide of the F72 fimbriae, as will be discussed.

L73 ANSWER 42 OF 56 HCAPLUS COPYRIGHT 2004 ACS on STN
AB The immature form of glycoprotein pgC purified from herpes simplex virus type 1-infected **ricin-resistant** (RicR14) cells carries an unusual sialylated glycopeptide that was sensitive to endo-β-N-acetylglucosaminidase. Gel filtration and polyacrylamide gel electrophoresis of pgC showed an N-linked **high-mannose** glycan and sialylated O-linked chain.

L73 ANSWER 43 OF 56 MEDLINE on STN DUPLICATE 24
AB The uropathogenic Escherichia coli KS52 strain expresses a **mannose-resistant hemagglutinin** involving an erythrocyte recognition site distinct from the alpha-digalactoside glycosphingolipid receptor identified for the uropathogenic E. coli strains specifying a P adhesin. The KS52 strain showed three major properties. (i) It agglutinated human erythrocytes of all tested blood groups. (ii) Hemagglutinin activity was found both in the supernatant fluid L-broth cultures and in cells grown on L-agar plates. (iii) No fimbriae in organisms grown on L-agar plates were detected by electron microscopy. Whole-cell DNA from the KS52 strain was size fractionated and cloned into the pHC79 cosmid vector. Three **recombinant** cosmids expressing a **mannose-resistant hemagglutination** (MRHA) phenotype were characterized and used to subclone the smallest DNA fragment able to confer the same MRHA properties as the parent strain. A 6.7-kilobase chromosomal DNA fragment cloned in pBR322 (pIL14) was shown to be necessary for host-cell MRHA expression and uroepithelial cell adherence. The insert encoded the production of a 16,000-dalton hemagglutinin. This polypeptide could be detected in culture supernatant fluids, in E. coli minicells harboring the pIL14 plasmid, and, by immunoblotting, in the KS52 strain and E. coli whole cells harboring the pIL14 plasmid. No homology was detected by Southern hybridization between the cloned insert and the DNA of the operon responsible for MRHA in the P-specifying, fimbriate strains (pap operon).

L73 ANSWER 44 OF 56 HCAPLUS COPYRIGHT 2004 ACS on STN
AB Unavailable

L73 ANSWER 45 OF 56 MEDLINE on STN DUPLICATE 25
AB In human fibroblasts, the receptor for low density lipoprotein (LDL) is

synthesized as a precursor of apparent Mr = 120,000 which is converted to a mature form of apparent Mr = 160,000, as determined by migration in sodium dodecyl sulfate (SDS)-polyacrylamide gels (Tolleshaug, H., Goldstein, J. L., Schneider, W. J., and Brown, M. S. (1982) Cell 30, 715-724). The current paper describes the relationship of N- and O-glycosylation to this post-translational modification. Oligosaccharides were analyzed from precursor and mature forms of LDL receptors that had been immunoprecipitated from cells grown in media containing radioactive sugars. In human epidermoid carcinoma A-431 cells, the receptor precursor appears to contain one N-linked **high mannose** oligosaccharide and approximately 6-9 N-acetylgalactosamine residues linked O-glycosidically to Ser/Thr residues. In the mature receptor, the O-linked oligosaccharides are mono- and disialylated species having the core structure of galactose leads to N-acetylgalactosamine leads to Ser/Thr. The single N-linked oligosaccharide of the mature receptor can either be a tri- or tetraantennary complex-type species. Similar results were obtained with normal human fibroblast receptor except that the O-linked oligosaccharides on the precursor are neutral disaccharides, of which one component is GalNAc and the N-linked complex type unit on the mature receptor is less branched. Since the addition of GalNAc residues to Ser/Thr residues precedes the conversion of N-linked **high mannose**-type oligosaccharides to complex-type structures, the transfer of N-acetylgalactosamine must occur prior to the entry of glycoproteins into the region of the Golgi containing the processing enzyme alpha-mannosidase I. We also studied the receptor from tunicamycin-treated cells and after treatment with neuraminidase. In addition, we analyzed the receptor synthesized by a **lectin-resistant** clone of Chinese hamster ovary cells that is deficient in adding galactose residues to both N- and O-linked oligosaccharides. These studies suggest that the apparent differences in molecular weight between the precursor and mature forms of the LDL receptor are largely, if not entirely, due to the addition of sialic acid and galactose residues to the O-linked GalNAc residues.(ABSTRACT TRUNCATED AT 400 WORDS)

L73 ANSWER 46 OF 56 MEDLINE on STN DUPLICATE 26

AB The O-linked oligosaccharides on mature forms of herpes simplex virus type 1 (HSV1) glycoproteins were characterized, and were found to account largely for the lower electrophoretic mobilities of these forms relative to the mobilities of immature forms. Other posttranslational modifications of HSV1 glycoproteins (designated gB, gC, gD and gE) were related temporally to the discrete shifts in electrophoretic mobilities that signal acquisition of the O-linked oligosaccharides. Fatty acid acylation (principally of gE) could be detected just prior to the shifts, whereas conversion of **high-mannose**-type N-linked oligosaccharides to the complex type occurred coincident with the shifts. The addition of O-linked oligosaccharides did not occur in cells treated with the ionophore monensin or in a **ricin-resistant** cell line defective in the processing of N-linked oligosaccharides. We conclude that extension of O-linked oligosaccharide chains on HSV1 glycoproteins, and probably also attachment of the first O-linked sugars, occurs as a late posttranslational modification in the Golgi apparatus.

L73 ANSWER 47 OF 56 MEDLINE on STN DUPLICATE 27

AB We report on N-acetylgalactosaminyltransferase (UDPacetylgalactosamine--protein acetylgalactosaminyltransferase; EC 2.4.1.41) activity in herpes simplex virus type 1 (HSV-1)-infected BHK and RicR14 cells, a line of **ricin-resistant** BHK cells defective in N-acetylglucosaminyltransferase I. The enzyme catalyzed the transfer of [14C]N-acetylgalactosamine (GalNAc) from UDP-[14C]GalNAc into HSV glycoproteins, as identified by immunoprecipitation. The sugar was selectively incorporated into the immature forms of herpesvirus glycoproteins pgC, pgD, and gA-pgB, which are known to contain N-linked glycans of the **high-mannose** type. The high incorporation of [14C]GalNAc into endogenous acceptors of HSV-1-infected

RicR14 cells was consistent with the accumulation of immature forms of HSV glycoproteins which occurs in these cells. Mild alkaline borohydride treatment of glycoproteins labeled via GalNAc transferase showed that the transferred GalNAc was O-linked and represented the first sugar added to the peptide backbone.

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AB We report on N-acetylgalactosaminyltransferase (UDPacetylgalactosamine-protein acetylgalactosaminyltransferase; EC 2.4.1.41) activity in herpes simplex virus type 1 (HSV-1)-infected BHK and Ric(R)14 cells, a line of **ricin-resistant** and BHK cells defective in N-acetylglucosaminyltransferase I. The enzyme catalyzed the transfer of [14C]N-acetylgalactosamine (GalNAc) from UDP-[14C]GalNAc into HSV glycoproteins, as identified by immunoprecipitation. The sugar was selectively incorporated into the immature forms of herpesvirus glycoproteins pgC, pgD, and gA-pgB, which are known to contain N-linked glycans of the **high-mannose** type. The **high** incorporation of [14C]GalNAc into endogenous acceptors of HSV-1-infected Ric(R)14 cells was consistent with the accumulation of immature forms of HSV glycoproteins which occurs in these cells. Mild alkaline borohydride treatment of glycoproteins labeled via GalNAc transferase showed that the transferred GalNAc was O-linked and represented the first sugar added to the peptide backbone.

L73 ANSWER 49 OF 56 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED. on STN DUPLICATE 28

AB Swainsonine is an indolizidine alkaloid that inhibits glycoprotein processing by inhibiting mannosidase II. Thus, cells grown in the presence of this alkaloid exhibit a decreased amount of complex types of oligosaccharides at their cell surface, and instead have hybrid types of structures. Since this compound could be useful for studying functional roles of glycoproteins, it was important to determine whether it affected the growth of mammalian cells in culture, and whether it was cytotoxic to these cells. At levels of up to 1 μg/ml, swainsonine did not affect the growth rate of Madin-Darbey canine kidney (MDCK) cells, Chinese hamster ovary (CHO), simian virus-181 (SV-101), B-16 melanoma, or intestine 407 cells, as measured by the increase in cell numbers over a 5-day period. There was also no apparent change in cell size or cell shape in cells grown in the presence of this inhibitor. Swainsonine also did not appear to be cytotoxic, nor to cause alterations in cell morphology, as evidenced by comparison of thin sections of normal and swainsonine-grown cells in the electron microscope. Since alterations in the oligosaccharide chains of cell surface glycoproteins could greatly affect cell surface properties, we examined the binding of various lectins and bacteria to cells grown in swainsonine as a measure of changes in their cell surface carbohydrates. Thus, when MDCK cells, CHO cells, or B-16 melanoma cells were grown for several days in the presence of swainsonine (100-500 ng/ml), these cells showed a 50-100% increase in their ability to bind [3H]concanavalin A, and a substantial decrease in the binding of [3H]wheat germ agglutinin. These alterations suggested an increase in **high -mannose** (or hybrid) types of receptors and a decrease in the complex types. The adhesion of E. coli B-886, a bacterium that binds to **high-mannose** glycoproteins, was also increased 1.5-to twofold, in cells grown in swainsonine. However, the binding of E. coli SS-142, another bacterial strain that does not bind to **high-mannose** receptors, was not altered by growth in swainsonine. In addition to the decrease in wheat germ agglutinin binding, another indication of a decrease in complex chains was the finding that CHO cells grown in swainsonine were more resistant to the toxic effects of the lectin, **ricin.** This increased **resistance** could be measured microscopically by the decrease in the number of cells remaining attached to the plates, or by the inhibition of amino acid incorporation, at various ricin concentrations. The effect of swainsonine in the

incorporation of amino acids and sugars into protein was also examined. When MDCK cells were grown overnight in swainsonine (1 μg/ml), or were incubated in the alkaloid for several hours before the start of the experiment, there was no alteration in the incorporation of [3H]leucine or [3H]proline into protein. There was, however, a significant inhibition in the incorporation of [3H]fucose, [3H]glucosamine, and [3H]galactose caused by this alkaloid. Fucose incorporation was decreased by about 40%, glucosamine by about 40 or 50%, and galactose by about 50%. In many cases (but not all), the incorporation of mannose was enhanced about 20-30% in cells grown in swainsonine.

L73 ANSWER 50 OF 56 HCAPLUS COPYRIGHT 2004 ACS on STN
AB DNA from E. coli strain AD110 was ligated to the vector plasmid pJB8 to form **recombinant** cosmids, which were packaged into phage λ particles and transduced into the E. coli K-12 strain JA221; when .apprx.800 ampicillin-resistant transductants were examined for **mannose-resistant hemagglutination** of human erythrocytes, 2 pos. colonies, each of which contained a plasmid (pPIL110-2 and pPIL110-3) in the expected size range [35-50 kilobases (kb)], were found. Aside from vector DNA, the 2 plasmids had only 1 EcoRI fragment, of 16 kb, in common. The 16-kb fragment was isolated, ligated to plasmid pACYC184 DNA, and transformed into JA221. Transformants that were tetracycline resistant and chloramphenicol sensitive caused the **mannose-resistant hemagglutination** of human erythrocytes. The restriction map of plasmid pPIL110-35 from one of these transformants was determined Strain JA221 cells containing pPIL110-35 showed a **mannose-resistant hemagglutination** capacity 4-8-fold that of AD110 cells. Plasmid pPIL110-35 encodes a fimbrial protein of mol. weight 17,000, as shown by SDS-polyacrylamide gel electrophoresis.

L73 ANSWER 51 OF 56 MEDLINE on STN DUPLICATE 29
AB Glycosylation of asparagine residues of glycoproteins occurs by the transfer of a glucose3mannose9N-acetylglucosamine2 (Glc3Man9GlcNAc2) oligosaccharide from a lipid carrier to the nascent protein. Normally, this transfer is quickly followed by the stepwise removal of the glucose residues which are arranged in the sequence: Glc1 leads to 2Glc1 leads to 3Glc1 leads to 3Man. We now report studies which demonstrate that a **lectin-resistant** mutant of the BW5147 mouse lymphoma cell line is deficient in the enzyme which removes the two inner glucose residues. This cell line (PHAR2.7) was selected for resistance to the cytotoxic effects of Phaseolus vulgaris leukoagglutinating lectin (Trowbridge, I. S., Hyman, R., Ferson, T., and Mazauskas, C. (1978) Eur. J. Immunol. 8, 716-723). Glycopeptides prepared from cells equilibrium-labeled with either [2-3H]mannose or [6-3H]galactose were characterized using lectin affinity chromatography, treatment with specific endo- and exoglycosidases, sizing by paper chromatography, and methylation analysis. Approximately 50% of the radioactivity in [3H]mannose-labeled glycopeptides from the mutant cells is present as glucosylated **high mannose**-type oligosaccharides whereas parent cell glycopeptides labeled under similar conditions lack detectable amounts of these species. Using [3H]galactose labeling, the major glucosylated oligosaccharides were identified as Glc2Man9GlcNAc2 and Glc2Man8GlcNAc2. In vitro enzyme assays demonstrated that the mutant cells cannot remove either of the two inner 1 leads to 3-linked glucose residues. Removal of the outer 1 leads to 3-linked glucose is normal. We conclude from these data that the PHAR2.7 cell line is deficient in glucosidase II, the enzyme which removes the two inner glucose residues from the oligosaccharides of newly glycosylated proteins.

L73 ANSWER 52 OF 56 MEDLINE on STN DUPLICATE 30
AB We have cloned the chromosomal hemolysin determinants from Escherichia coli strains belonging to the four O-serotypes O4, O6, O18, and O75. The hemolysin-producing clones were isolated from gene banks of these strains

which were constructed by inserting partial Sau3A fragments of chromosomal DNA into the cosmid pJC74. The hemolytic cosmid clones were relatively stable. The inserts were further subcloned either as SalI fragments in pACYC184 or as BamHI-SalI fragments in a **recombinant** plasmid (pANN202) containing cistron C (hlyC) of the plasmid-encoded hemolysin determinant. Detailed restriction maps of each of these determinants were constructed, and it was found that, despite sharing overall homology, the determinants exhibited minor specific differences in their structure. These appeared to be restricted to cistron A (hlyA), which is the structural gene for hemolysin. In the gene banks of two of these hemolytic strains, we could also identify clones which carried the genetic determinants for the **mannose-resistant hemagglutination** antigens Vb and VIc. Both of these fimbrial antigens were expressed in the E. coli K-12 clones to an extent similar to that observed in the wild-type strains. These **recombinant** cosmids were rather unstable, and, in the absence of selection, segregated at a high frequency.

L73 ANSWER 53 OF 56 MEDLINE on STN DUPLICATE 31
AB Chromosomal DNA from a uropathogenic strain of Escherichia coli was partially digested with the restriction enzyme EcoRI. The partial digests were ligated into a cosmid containing an ampicillin-resistant determinant and packaged into lambda phage particles. An ampicillin-resistant transductant of E. coli HB101 was found to possess **mannose-resistant hemagglutinating** activity associated with a 50-kilobase-pair plasmid. Subcloning of the **mannose**-resistant fimbrial genes revealed that the genetic determinants were encoded by a 6.9-kilobase-pair DNA fragment of a **recombinant** plasmid. Chimeric plasmids smaller in size were unable to transform E. coli to fimbrial production. Physical maps of the **recombinant** plasmids were prepared showing restriction endonuclease sites within the inserted DNA fragments. The hemagglutinating activities of the wild-type strain and of the **recombinant** derivative were compared. Both strains agglutinated human erythrocytes in the presence of D-**mannose** to the same degree and also failed to produce fimbriae after incubation at 18 degrees C. Also, both strains were agglutinated by antifimbrial serum at a high titer, whereas no such activity was observed when a strain of E. coli which did not possess a plasmid was used.

L73 ANSWER 54 OF 56 MEDLINE on STN
AB We have developed a new procedure for introducing macromolecules into cultured mammalian cells based on osmotic lysis of pinocytic vesicles. Cells are first incubated in culture medium containing 0.5 M sucrose, 10% polyethylene glycol 1000 and the macromolecule to be transferred. Cells are then placed in medium diluted with 0.66 parts water. Most pinocytic vesicles formed in the presence of sucrose burst in hypotonic medium, thereby releasing the enclosed macromolecule. L929 cells remain fully viable after a single hypertonic sucrose treatment, and a majority survives four successive rounds of osmotic lysis. This procedure, termed osmotic lysis of pinosomes, has been used to transfer substantial amounts of horseradish peroxidase, antiricin antibodies and dextran 70,000 into the cytosol of L929 cells. Direct comparison of the degree of **ricin resistance** conferred by transfer of antiricin antibodies revealed pinosome lysis to be equal, if not superior, to injection mediated by red blood cells.

L73 ANSWER 55 OF 56 MEDLINE on STN DUPLICATE 32
AB Isolates of Escherichia coli from human urinary tract infections frequently express adherence properties found less often among normal intestinal isolates. These properties include adherence to human uroepithelial cells and primary monkey kidney cells, as well as D-**mannose-resistant hemagglutination** of human erythrocytes, and they are mediated by a pilus type different from type 1. The genes encoding this pilus type (pyelonephritis-associated pili, pap)

and those encoding type 1 pili have been cloned from a urinary tract infection isolate of E. coli and transferred to an E. coli K-12 derivative. The **recombinant** plasmids were found to express functional pili and to endow the new host with all of the adherence properties of the urinary tract infection isolate. Both pilus types were found to be genetically distinct, and unlike the adherence genes from bovine, porcine, and human diarrheal isolates, both were found to be chromosomally encoded.

L73 ANSWER 56 OF 56 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AB The K99 antigen common to some bovine strains of E. coli caused **mannose-resistant hemagglutination** of sheep erythrocytes; it was apparently responsible for the attachment of K99-positive bacteria to calf brush-border preparations as strains grown at 18° C did not produce K99 antigen, cause hemagglutination or attach to brush borders. Also, a K12 (K99+) **recombinant** strain showed hemagglutinating activity and attachment to brush borders; before it received the K99 plasmid, the recipient strain was negative in both respects. Cell-free extracts of K99 antigen showed hemagglutinating activity and inhibited the attachment of K99-positive organisms to brush borders. K99 antigen appears to be a virulence determinant in the pathogenesis of neonatal calf diarrhea. It is readily demonstrated by hemagglutination and brush border attachment tests.

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COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
                                                         ENTRY    SESSION
FULL ESTIMATED COST                                       0.63       0.63

FILES 'MEDLINE, SCISEARCH, LIFESCI, BIOTECHDS, BIOSIS, EMBASE, HCAPLUS, NTIS,
       ESBIOBASE, BIOTECHNO, WPIDS' ENTERED AT 15:27:54 ON 01 JUN 2004
ALL COPYRIGHTS AND RESTRICTIONS APPLY.  SEE HELP USAGETERMS FOR DETAILS.

11 FILES IN THE FILE LIST

=> s alpha glucosidase#
FILE 'MEDLINE'
        477015 ALPHA
         10338 GLUCOSIDASE#
L1        4376 ALPHA GLUCOSIDASE#
                (ALPHA(W)GLUCOSIDASE#)

FILE 'SCISEARCH'
        677809 ALPHA
          8097 GLUCOSIDASE#
L2        2672 ALPHA GLUCOSIDASE#
                (ALPHA(W)GLUCOSIDASE#)
```

```
FILE 'LIFESCI'
        153327 "ALPHA"
          3975 GLUCOSIDASE#
L3        1459 ALPHA GLUCOSIDASE#
                 ("ALPHA"(W)GLUCOSIDASE#)

FILE 'BIOTECHDS'
         25989 ALPHA
          3114 GLUCOSIDASE#
L4         656 ALPHA GLUCOSIDASE#
                 (ALPHA(W)GLUCOSIDASE#)

FILE 'BIOSIS'
        623794 ALPHA
         10929 GLUCOSIDASE#
L5        3716 ALPHA GLUCOSIDASE#
                 (ALPHA(W)GLUCOSIDASE#)

FILE 'EMBASE'
        536116 "ALPHA"
          9325 GLUCOSIDASE#
L6        5092 ALPHA GLUCOSIDASE#
                 ("ALPHA"(W)GLUCOSIDASE#)

FILE 'HCAPLUS'
       1476958 ALPHA
         16114 GLUCOSIDASE#
L7        5264 ALPHA GLUCOSIDASE#
                 (ALPHA(W)GLUCOSIDASE#)

FILE 'NTIS'
         28579 ALPHA
            91 GLUCOSIDASE#
L8           5 ALPHA GLUCOSIDASE#
                 (ALPHA(W)GLUCOSIDASE#)

FILE 'ESBIOBASE'
        192999 ALPHA
          5331 GLUCOSIDASE#
L9         784 ALPHA GLUCOSIDASE#
                 (ALPHA(W)GLUCOSIDASE#)

FILE 'BIOTECHNO'
        189431 ALPHA
          4274 GLUCOSIDASE#
L10       1799 ALPHA GLUCOSIDASE#
                 (ALPHA(W)GLUCOSIDASE#)

FILE 'WPIDS'
        174250 ALPHA
          1547 GLUCOSIDASE#
L11        687 ALPHA GLUCOSIDASE#
                 (ALPHA(W)GLUCOSIDASE#)

TOTAL FOR ALL FILES
L12      26510 ALPHA GLUCOSIDASE#

=> s l12 and high mannose
FILE 'MEDLINE'
       1212315 HIGH
         17284 MANNOSE
          1942 HIGH MANNOSE
                 (HIGH(W)MANNOSE)
L13         53 L1 AND HIGH MANNOSE
```

```
FILE 'SCISEARCH'
       1767795 HIGH
         12985 MANNOSE
          1278 HIGH MANNOSE
                 (HIGH(W)MANNOSE)
L14          10 L2 AND HIGH MANNOSE

FILE 'LIFESCI'
        333161 "HIGH"
          5734 "MANNOSE"
           631 HIGH MANNOSE
                 ("HIGH"(W)"MANNOSE")
L15           9 L3 AND HIGH MANNOSE

FILE 'BIOTECHDS'
         62833 HIGH
          1672 MANNOSE
           136 HIGH MANNOSE
                 (HIGH(W)MANNOSE)
L16           5 L4 AND HIGH MANNOSE

FILE 'BIOSIS'
       1349013 HIGH
         19914 MANNOSE
          2052 HIGH MANNOSE
                 (HIGH(W)MANNOSE)
L17          18 L5 AND HIGH MANNOSE

FILE 'EMBASE'
       1168335 "HIGH"
         13513 "MANNOSE"
          1600 HIGH MANNOSE
                 ("HIGH"(W)"MANNOSE")
L18          26 L6 AND HIGH MANNOSE

FILE 'HCAPLUS'
       3362832 HIGH
         35664 MANNOSE
          2292 HIGH MANNOSE
                 (HIGH(W)MANNOSE)
L19          28 L7 AND HIGH MANNOSE

FILE 'NTIS'
        319287 HIGH
           112 MANNOSE
             6 HIGH MANNOSE
                 (HIGH(W)MANNOSE)
L20           0 L8 AND HIGH MANNOSE

FILE 'ESBIOBASE'
        413248 HIGH
          5153 MANNOSE
           612 HIGH MANNOSE
                 (HIGH(W)MANNOSE)
L21           4 L9 AND HIGH MANNOSE

FILE 'BIOTECHNO'
        299126 HIGH
          7168 MANNOSE
          1188 HIGH MANNOSE
                 (HIGH(W)MANNOSE)
L22          19 L10 AND HIGH MANNOSE
```

```
FILE 'WPIDS'
       1831128 HIGH
          2583 MANNOSE
            52 HIGH MANNOSE
                 (HIGH(W)MANNOSE)
L23           4 L11 AND HIGH MANNOSE

TOTAL FOR ALL FILES
L24         176 L12 AND HIGH MANNOSE

=> s l24 not 2002-2004/py
FILE 'MEDLINE'
       1314032 2002-2004/PY
L25          49 L13 NOT 2002-2004/PY

FILE 'SCISEARCH'
       2399042 2002-2004/PY
L26           8 L14 NOT 2002-2004/PY

FILE 'LIFESCI'
        214244 2002-2004/PY
L27           9 L15 NOT 2002-2004/PY

FILE 'BIOTECHDS'
         53433 2002-2004/PY
L28           1 L16 NOT 2002-2004/PY

FILE 'BIOSIS'
       1231965 2002-2004/PY
L29          16 L17 NOT 2002-2004/PY

FILE 'EMBASE'
       1099415 2002-2004/PY
L30          25 L18 NOT 2002-2004/PY

FILE 'HCAPLUS'
       2499762 2002-2004/PY
L31          23 L19 NOT 2002-2004/PY

FILE 'NTIS'
         27941 2002-2004/PY
L32           0 L20 NOT 2002-2004/PY

FILE 'ESBIOBASE'
        683260 2002-2004/PY
L33           2 L21 NOT 2002-2004/PY

FILE 'BIOTECHNO'
        244553 2002-2004/PY
L34          18 L22 NOT 2002-2004/PY

FILE 'WPIDS'
       2484316 2002-2004/PY
L35           0 L23 NOT 2002-2004/PY

TOTAL FOR ALL FILES
L36         151 L24 NOT 2002-2004/PY

=> s lysosom? and high mannose
FILE 'MEDLINE'
         39021 LYSOSOM?
       1212315 HIGH
         17284 MANNOSE
          1942 HIGH MANNOSE
```

```
                (HIGH(W)MANNOSE)
L37         181 LYSOSOM? AND HIGH MANNOSE

FILE 'SCISEARCH'
          22909 LYSOSOM?
        1767795 HIGH
          12985 MANNOSE
           1278 HIGH MANNOSE
                (HIGH(W)MANNOSE)
L38         102 LYSOSOM? AND HIGH MANNOSE

FILE 'LIFESCI'
           7194 LYSOSOM?
         333161 "HIGH"
           5734 "MANNOSE"
            631 HIGH MANNOSE
                ("HIGH"(W)"MANNOSE")
L39          32 LYSOSOM? AND HIGH MANNOSE

FILE 'BIOTECHDS'
            488 LYSOSOM?
          62833 HIGH
           1672 MANNOSE
            136 HIGH MANNOSE
                (HIGH(W)MANNOSE)
L40           6 LYSOSOM? AND HIGH MANNOSE

FILE 'BIOSIS'
          38506 LYSOSOM?
        1349013 HIGH
          19914 MANNOSE
           2052 HIGH MANNOSE
                (HIGH(W)MANNOSE)
L41         176 LYSOSOM? AND HIGH MANNOSE

FILE 'EMBASE'
          30316 LYSOSOM?
        1168335 "HIGH"
          13513 "MANNOSE"
           1600 HIGH MANNOSE
                ("HIGH"(W)"MANNOSE")
L42         133 LYSOSOM? AND HIGH MANNOSE

FILE 'HCAPLUS'
          33872 LYSOSOM?
        3362832 HIGH
          35664 MANNOSE
           2292 HIGH MANNOSE
                (HIGH(W)MANNOSE)
L43         188 LYSOSOM? AND HIGH MANNOSE

FILE 'NTIS'
            280 LYSOSOM?
         319287 HIGH
            112 MANNOSE
              6 HIGH MANNOSE
                (HIGH(W)MANNOSE)
L44           1 LYSOSOM? AND HIGH MANNOSE

FILE 'ESBIOBASE'
           8550 LYSOSOM?
         413248 HIGH
           5153 MANNOSE
            612 HIGH MANNOSE
```

```
                (HIGH(W)MANNOSE)
L45         49 LYSOSOM? AND HIGH MANNOSE

FILE 'BIOTECHNO'
          8722 LYSOSOM?
        299126 HIGH
          7168 MANNOSE
          1188 HIGH MANNOSE
                (HIGH(W)MANNOSE)
L46         100 LYSOSOM? AND HIGH MANNOSE

FILE 'WPIDS'
           600 LYSOSOM?
       1831128 HIGH
          2583 MANNOSE
            52 HIGH MANNOSE
                (HIGH(W)MANNOSE)
L47           6 LYSOSOM? AND HIGH MANNOSE

TOTAL FOR ALL FILES
L48         974 LYSOSOM? AND HIGH MANNOSE

=> s l48 and (gaa or acid glucosidase#)
FILE 'MEDLINE'
           771 GAA
       1262731 ACID
         10338 GLUCOSIDASE#
            21 ACID GLUCOSIDASE#
                (ACID(W)GLUCOSIDASE#)
L49           0 L37 AND (GAA OR ACID GLUCOSIDASE#)

FILE 'SCISEARCH'
           829 GAA
        984260 ACID
          8097 GLUCOSIDASE#
            11 ACID GLUCOSIDASE#
                (ACID(W)GLUCOSIDASE#)
L50           0 L38 AND (GAA OR ACID GLUCOSIDASE#)

FILE 'LIFESCI'
           348 GAA
        273037 "ACID"
          3975 GLUCOSIDASE#
             0 ACID GLUCOSIDASE#
                ("ACID"(W)GLUCOSIDASE#)
L51           0 L39 AND (GAA OR ACID GLUCOSIDASE#)

FILE 'BIOTECHDS'
           173 GAA
        113452 ACID
          3114 GLUCOSIDASE#
             0 ACID GLUCOSIDASE#
                (ACID(W)GLUCOSIDASE#)
L52           0 L40 AND (GAA OR ACID GLUCOSIDASE#)

FILE 'BIOSIS'
           844 GAA
       1157942 ACID
         10929 GLUCOSIDASE#
            17 ACID GLUCOSIDASE#
                (ACID(W)GLUCOSIDASE#)
L53           0 L41 AND (GAA OR ACID GLUCOSIDASE#)

FILE 'EMBASE'
```

```
          652 GAA
      1223343 "ACID"
         9325 GLUCOSIDASE#
           15 ACID GLUCOSIDASE#
                ("ACID"(W)GLUCOSIDASE#)
L54           0 L42 AND (GAA OR ACID GLUCOSIDASE#)

FILE 'HCAPLUS'
         1139 GAA
      3823737 ACID
        16114 GLUCOSIDASE#
          126 ACID GLUCOSIDASE#
                (ACID(W)GLUCOSIDASE#)
L55           0 L43 AND (GAA OR ACID GLUCOSIDASE#)

FILE 'NTIS'
          158 GAA
        43272 ACID
           91 GLUCOSIDASE#
            0 ACID GLUCOSIDASE#
                (ACID(W)GLUCOSIDASE#)
L56           0 L44 AND (GAA OR ACID GLUCOSIDASE#)

FILE 'ESBIOBASE'
          457 GAA
       283047 ACID
         5331 GLUCOSIDASE#
            8 ACID GLUCOSIDASE#
                (ACID(W)GLUCOSIDASE#)
L57           0 L45 AND (GAA OR ACID GLUCOSIDASE#)

FILE 'BIOTECHNO'
          448 GAA
       349810 ACID
         4274 GLUCOSIDASE#
            0 ACID GLUCOSIDASE#
                (ACID(W)GLUCOSIDASE#)
L58           0 L46 AND (GAA OR ACID GLUCOSIDASE#)

FILE 'WPIDS'
          209 GAA
       847217 ACID
         1547 GLUCOSIDASE#
            0 ACID GLUCOSIDASE#
                (ACID(W)GLUCOSIDASE#)
L59           0 L47 AND (GAA OR ACID GLUCOSIDASE#)

TOTAL FOR ALL FILES
L60           0 L48 AND (GAA OR ACID GLUCOSIDASE#)

=> s l48 and glucosidase#
FILE 'MEDLINE'
        10338 GLUCOSIDASE#
L61        16 L37 AND GLUCOSIDASE#

FILE 'SCISEARCH'
         8097 GLUCOSIDASE#
L62         7 L38 AND GLUCOSIDASE#

FILE 'LIFESCI'
         3975 GLUCOSIDASE#
L63         1 L39 AND GLUCOSIDASE#

FILE 'BIOTECHDS'
```

```
          3114 GLUCOSIDASE#
L64          3 L40 AND GLUCOSIDASE#

FILE 'BIOSIS'
         10929 GLUCOSIDASE#
L65         11 L41 AND GLUCOSIDASE#

FILE 'EMBASE'
          9325 GLUCOSIDASE#
L66          8 L42 AND GLUCOSIDASE#

FILE 'HCAPLUS'
         16114 GLUCOSIDASE#
L67         13 L43 AND GLUCOSIDASE#

FILE 'NTIS'
            91 GLUCOSIDASE#
L68          0 L44 AND GLUCOSIDASE#

FILE 'ESBIOBASE'
          5331 GLUCOSIDASE#
L69         10 L45 AND GLUCOSIDASE#

FILE 'BIOTECHNO'
          4274 GLUCOSIDASE#
L70          7 L46 AND GLUCOSIDASE#

FILE 'WPIDS'
          1547 GLUCOSIDASE#
L71          4 L47 AND GLUCOSIDASE#

TOTAL FOR ALL FILES
L72         80 L48 AND GLUCOSIDASE#

=> dup rem l72
PROCESSING COMPLETED FOR L72
L73          35 DUP REM L72 (45 DUPLICATES REMOVED)

=> d

L73   ANSWER 1 OF 35  BIOTECHDS COPYRIGHT 2004 THOMSON DERWENT/ISI on STN
TI    Producing a glycoprotein with reduced complex carbohydrates by culturing
      the lectin resistant mammalian cell expressing the glycoprotein for
      treating lysosomal storage disease;
         recombinant protein production via host cell culture for use in
         disease therapy and gene therapy
AU    CANFIELD W M
AN    2004-00315  BIOTECHDS
PI    US 2003124653 3 Jul 2003


=> s alpha glycosidase#
FILE 'MEDLINE'
        477015 ALPHA
          4557 GLYCOSIDASE#
L74         40 ALPHA GLYCOSIDASE#
                  (ALPHA(W)GLYCOSIDASE#)

FILE 'SCISEARCH'
        677809 ALPHA
          5031 GLYCOSIDASE#
L75         42 ALPHA GLYCOSIDASE#
                  (ALPHA(W)GLYCOSIDASE#)
```

```
FILE 'LIFESCI'
        153327 "ALPHA"
          1404 GLYCOSIDASE#
L76         16 ALPHA GLYCOSIDASE#
                  ("ALPHA"(W)GLYCOSIDASE#)

FILE 'BIOTECHDS'
         25989 ALPHA
           568 GLYCOSIDASE#
L77         10 ALPHA GLYCOSIDASE#
                  (ALPHA(W)GLYCOSIDASE#)

FILE 'BIOSIS'
        623794 ALPHA
          5742 GLYCOSIDASE#
L78         90 ALPHA GLYCOSIDASE#
                  (ALPHA(W)GLYCOSIDASE#)

FILE 'EMBASE'
        536116 "ALPHA"
          4915 GLYCOSIDASE#
L79         55 ALPHA GLYCOSIDASE#
                  ("ALPHA"(W)GLYCOSIDASE#)

FILE 'HCAPLUS'
       1476958 ALPHA
          7619 GLYCOSIDASE#
L80        184 ALPHA GLYCOSIDASE#
                  (ALPHA(W)GLYCOSIDASE#)

FILE 'NTIS'
         28579 ALPHA
            49 GLYCOSIDASE#
L81          1 ALPHA GLYCOSIDASE#
                  (ALPHA(W)GLYCOSIDASE#)

FILE 'ESBIOBASE'
        192999 ALPHA
          1750 GLYCOSIDASE#
L82         11 ALPHA GLYCOSIDASE#
                  (ALPHA(W)GLYCOSIDASE#)

FILE 'BIOTECHNO'
        189431 ALPHA
          2667 GLYCOSIDASE#
L83         13 ALPHA GLYCOSIDASE#
                  (ALPHA(W)GLYCOSIDASE#)

FILE 'WPIDS'
        174250 ALPHA
           596 GLYCOSIDASE#
L84         54 ALPHA GLYCOSIDASE#
                  (ALPHA(W)GLYCOSIDASE#)

TOTAL FOR ALL FILES
L85        516 ALPHA GLYCOSIDASE#

=> s l85 and high mannose
FILE 'MEDLINE'
       1212315 HIGH
         17284 MANNOSE
          1942 HIGH MANNOSE
                  (HIGH(W)MANNOSE)
L86          0 L74 AND HIGH MANNOSE
```

```
FILE 'SCISEARCH'
       1767795 HIGH
         12985 MANNOSE
          1278 HIGH MANNOSE
                 (HIGH(W)MANNOSE)
L87           0 L75 AND HIGH MANNOSE

FILE 'LIFESCI'
        333161 "HIGH"
          5734 "MANNOSE"
           631 HIGH MANNOSE
                 ("HIGH"(W)"MANNOSE")
L88           0 L76 AND HIGH MANNOSE

FILE 'BIOTECHDS'
         62833 HIGH
          1672 MANNOSE
           136 HIGH MANNOSE
                 (HIGH(W)MANNOSE)
L89           1 L77 AND HIGH MANNOSE

FILE 'BIOSIS'
       1349013 HIGH
         19914 MANNOSE
          2052 HIGH MANNOSE
                 (HIGH(W)MANNOSE)
L90           0 L78 AND HIGH MANNOSE

FILE 'EMBASE'
       1168335 "HIGH"
         13513 "MANNOSE"
          1600 HIGH MANNOSE
                 ("HIGH"(W)"MANNOSE")
L91           0 L79 AND HIGH MANNOSE

FILE 'HCAPLUS'
       3362832 HIGH
         35664 MANNOSE
          2292 HIGH MANNOSE
                 (HIGH(W)MANNOSE)
L92           0 L80 AND HIGH MANNOSE

FILE 'NTIS'
        319287 HIGH
           112 MANNOSE
             6 HIGH MANNOSE
                 (HIGH(W)MANNOSE)
L93           0 L81 AND HIGH MANNOSE

FILE 'ESBIOBASE'
        413248 HIGH
          5153 MANNOSE
           612 HIGH MANNOSE
                 (HIGH(W)MANNOSE)
L94           0 L82 AND HIGH MANNOSE

FILE 'BIOTECHNO'
        299126 HIGH
          7168 MANNOSE
          1188 HIGH MANNOSE
                 (HIGH(W)MANNOSE)
L95           0 L83 AND HIGH MANNOSE
```

```
FILE 'WPIDS'
       1831128 HIGH
          2583 MANNOSE
            52 HIGH MANNOSE
                 (HIGH(W)MANNOSE)
L96          1 L84 AND HIGH MANNOSE

TOTAL FOR ALL FILES
L97          2 L85 AND HIGH MANNOSE

=> s lysosomal hydrolase#
FILE 'MEDLINE'
         21235 LYSOSOMAL
         70831 HYDROLASE#
L98       1046 LYSOSOMAL HYDROLASE#
                 (LYSOSOMAL(W)HYDROLASE#)

FILE 'SCISEARCH'
         15951 LYSOSOMAL
         15058 HYDROLASE#
L99        639 LYSOSOMAL HYDROLASE#
                 (LYSOSOMAL(W)HYDROLASE#)

FILE 'LIFESCI'
          4787 "LYSOSOMAL"
          4922 HYDROLASE#
L100       177 LYSOSOMAL HYDROLASE#
                 ("LYSOSOMAL"(W)HYDROLASE#)

FILE 'BIOTECHDS'
           367 LYSOSOMAL
          2236 HYDROLASE#
L101        14 LYSOSOMAL HYDROLASE#
                 (LYSOSOMAL(W)HYDROLASE#)

FILE 'BIOSIS'
         24759 LYSOSOMAL
         21501 HYDROLASE#
L102      1264 LYSOSOMAL HYDROLASE#
                 (LYSOSOMAL(W)HYDROLASE#)

FILE 'EMBASE'
         18831 "LYSOSOMAL"
         14666 HYDROLASE#
L103       990 LYSOSOMAL HYDROLASE#
                 ("LYSOSOMAL"(W)HYDROLASE#)

FILE 'HCAPLUS'
         22481 LYSOSOMAL
         22418 HYDROLASE#
L104      1129 LYSOSOMAL HYDROLASE#
                 (LYSOSOMAL(W)HYDROLASE#)

FILE 'NTIS'
           161 LYSOSOMAL
          1152 HYDROLASE#
L105         6 LYSOSOMAL HYDROLASE#
                 (LYSOSOMAL(W)HYDROLASE#)

FILE 'ESBIOBASE'
          5401 LYSOSOMAL
          5397 HYDROLASE#
L106       198 LYSOSOMAL HYDROLASE#
                 (LYSOSOMAL(W)HYDROLASE#)
```

```
FILE 'BIOTECHNO'
          6000 LYSOSOMAL
          6822 HYDROLASE#
L107       261 LYSOSOMAL HYDROLASE#
                 (LYSOSOMAL(W)HYDROLASE#)

FILE 'WPIDS'
           404 LYSOSOMAL
          2068 HYDROLASE#
L108        12 LYSOSOMAL HYDROLASE#
                 (LYSOSOMAL(W)HYDROLASE#)

TOTAL FOR ALL FILES
L109      5736 LYSOSOMAL HYDROLASE#

=> s l109 and mannose
FILE 'MEDLINE'
         17284 MANNOSE
L110        94 L98 AND MANNOSE

FILE 'SCISEARCH'
         12985 MANNOSE
L111        63 L99 AND MANNOSE

FILE 'LIFESCI'
          5734 MANNOSE
L112        20 L100 AND MANNOSE

FILE 'BIOTECHDS'
          1672 MANNOSE
L113         6 L101 AND MANNOSE

FILE 'BIOSIS'
         19914 MANNOSE
L114       101 L102 AND MANNOSE

FILE 'EMBASE'
         13513 MANNOSE
L115        78 L103 AND MANNOSE

FILE 'HCAPLUS'
         35664 MANNOSE
L116        96 L104 AND MANNOSE

FILE 'NTIS'
           112 MANNOSE
L117         0 L105 AND MANNOSE

FILE 'ESBIOBASE'
          5153 MANNOSE
L118        35 L106 AND MANNOSE

FILE 'BIOTECHNO'
          7168 MANNOSE
L119        46 L107 AND MANNOSE

FILE 'WPIDS'
          2583 MANNOSE
L120         5 L108 AND MANNOSE

TOTAL FOR ALL FILES
L121       544 L109 AND MANNOSE
```

```
=> s l121 and recombinant
FILE 'MEDLINE'
        224036 RECOMBINANT
L122         14 L110 AND RECOMBINANT

FILE 'SCISEARCH'
        132878 RECOMBINANT
L123          7 L111 AND RECOMBINANT

FILE 'LIFESCI'
         60086 RECOMBINANT
L124          2 L112 AND RECOMBINANT

FILE 'BIOTECHDS'
         81313 RECOMBINANT
L125          5 L113 AND RECOMBINANT

FILE 'BIOSIS'
        173817 RECOMBINANT
L126          5 L114 AND RECOMBINANT

FILE 'EMBASE'
        146461 RECOMBINANT
L127          3 L115 AND RECOMBINANT

FILE 'HCAPLUS'
        157184 RECOMBINANT
L128          6 L116 AND RECOMBINANT

FILE 'NTIS'
          1506 RECOMBINANT
L129          0 L117 AND RECOMBINANT

FILE 'ESBIOBASE'
         70828 RECOMBINANT
L130          4 L118 AND RECOMBINANT

FILE 'BIOTECHNO'
        125134 RECOMBINANT
L131          5 L119 AND RECOMBINANT

FILE 'WPIDS'
         34715 RECOMBINANT
L132          2 L120 AND RECOMBINANT

TOTAL FOR ALL FILES
L133         53 L121 AND RECOMBINANT

=> dup rem l133
PROCESSING COMPLETED FOR L133
L134         22 DUP REM L133 (31 DUPLICATES REMOVED)

=> d

L134  ANSWER 1 OF 22  BIOTECHDS COPYRIGHT 2004 THOMSON DERWENT/ISI on STN
TI    Phosphorylating a protein for treating a patient suffering from a
      lysosomal storage disease e.g. Fabry's disease by contacting the protein
      with a soluble GlcNAc-phosphotransferase and producing a phosphorylated
      protein;
           recombinant protein production for use in disease therapy
         and gene therapy
AU    CANFIELD W; KUDO M
AN    2003-28789  BIOTECHDS
PI    US 2003119088 26 Jun 2003
```

=> d 2-5

L134 ANSWER 2 OF 22 BIOTECHDS COPYRIGHT 2004 THOMSON DERWENT/ISI on STN
TI Producing a glycoprotein with reduced complex carbohydrates by culturing the lectin resistant mammalian cell expressing the glycoprotein for treating lysosomal storage disease;
recombinant protein production via host cell culture for use in disease therapy and gene therapy
AU CANFIELD W M
AN 2004-00315 BIOTECHDS
PI US 2003124653 3 Jul 2003

L134 ANSWER 3 OF 22 MEDLINE on STN
TI Phosphoregulation of sorting signal-VHS domain interactions by a direct electrostatic mechanism.
SO Nature structural biology, (2002 Jul) 9 (7) 532-6.
Journal code: 9421566. ISSN: 1072-8368.
AU Kato Yukio; Misra Saurav; Puertollano Rosa; Hurley James H; Bonifacino Juan S
AN 2002337810 MEDLINE

L134 ANSWER 4 OF 22 BIOTECHDS COPYRIGHT 2004 THOMSON DERWENT/ISI on STN
TI Novel N-acetylglucosamine-1-phosphotransferase and N-acetylglucosamine-1-phosphodiester-alpha-N-acetylglucosaminidase, useful for producing phosphorylated **lysosomal hydrolase** for treating lysosomal storage diseases;
vector-mediated gene transfer and expression in host cell, monoclonal antibody and hybridoma
AU Canfield W M
AN 2001-09921 BIOTECHDS
PI WO 2001019955 22 Mar 2001

L134 ANSWER 5 OF 22 MEDLINE on STN
TI **Lysosomal hydrolase mannose** 6-phosphate uncovering enzyme resides in the trans-Golgi network.
SO Molecular biology of the cell, (2001 Jun) 12 (6) 1623-31.
Journal code: 9201390. ISSN: 1059-1524.
AU Rohrer J; Kornfeld R
AN 2001345639 MEDLINE

=> s l121 not 2002-2004/py
FILE 'MEDLINE'
1314032 2002-2004/PY
L135 89 L110 NOT 2002-2004/PY

FILE 'SCISEARCH'
2399042 2002-2004/PY
L136 58 L111 NOT 2002-2004/PY

FILE 'LIFESCI'
214244 2002-2004/PY
L137 18 L112 NOT 2002-2004/PY

FILE 'BIOTECHDS'
53433 2002-2004/PY
L138 3 L113 NOT 2002-2004/PY

FILE 'BIOSIS'
1231965 2002-2004/PY
L139 95 L114 NOT 2002-2004/PY

FILE 'EMBASE'
1099415 2002-2004/PY
L140 74 L115 NOT 2002-2004/PY

FILE 'HCAPLUS'
2499762 2002-2004/PY
L141 89 L116 NOT 2002-2004/PY

FILE 'NTIS'
27941 2002-2004/PY
L142 0 L117 NOT 2002-2004/PY

FILE 'ESBIOBASE'
683260 2002-2004/PY
L143 31 L118 NOT 2002-2004/PY

FILE 'BIOTECHNO'
244553 2002-2004/PY
L144 44 L119 NOT 2002-2004/PY

FILE 'WPIDS'
2484316 2002-2004/PY
L145 0 L120 NOT 2002-2004/PY

TOTAL FOR ALL FILES
L146 501 L121 NOT 2002-2004/PY

=> dup rem l146
PROCESSING COMPLETED FOR L146
L147 141 DUP REM L146 (360 DUPLICATES REMOVED)

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L147 ANSWER 1 OF 141 BIOTECHDS COPYRIGHT 2004 THOMSON DERWENT/ISI on STN
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L147 ANSWER 48 OF 141 MEDLINE on STN
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L147 ANSWER 56 OF 141 MEDLINE on STN DUPLICATE 42
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L147 ANSWER 59 OF 141 MEDLINE on STN DUPLICATE 44
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AN 1980:245459 BIOSIS

L147 ANSWER 124 OF 141 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
DUPLICATE 85
TI BINDING OF **LYSOSOMAL HYDROLASES** BY THE MEMBRANES OF RAT LIVER LYSOSOMES EVIDENCE FOR PHOSPHO MANNOSYL RECOGNITION MARKERS.
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AN 1980:277495 BIOSIS

L147 ANSWER 125 OF 141 MEDLINE on STN DUPLICATE 86
TI The role of extra-hepatic tissues in the receptor-mediated plasma clearance of glycoproteins terminated by **mannose** or N-acetylglucosamine.
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Journal code: 2984726R. ISSN: 0264-6021.
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AN 81207182 MEDLINE

L147 ANSWER 126 OF 141 MEDLINE on STN DUPLICATE 87
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Journal code: 0217513. ISSN: 0006-3002.
AU Chien S F; Dawson G
AN 81000541 MEDLINE

L147 ANSWER 127 OF 141 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
DUPLICATE 88
TI INTER CELLULAR EXCHANGE OF **LYSOSOMAL HYDROLASES**
BETWEEN MUTANT HUMAN FIBROBLASTS AND OTHER CELL TYPES.
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CODEN: ECREAL. ISSN: 0014-4827.
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AN 1981:180907 BIOSIS

L147 ANSWER 128 OF 141 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
DUPLICATE 89
TI RICIN LINKED TO MONO PHOSPHO PENTA **MANNOSE** BINDS TO FIBROBLAST
LYSOSOMAL HYDROLASE RECEPTORS RESULTING IN A CELL TYPE
SPECIFIC TOXIN.
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L147 ANSWER 129 OF 141 MEDLINE on STN
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SO Proceedings of the National Academy of Sciences of the United States of America, (1979 Sep) 76 (9) 4322-6.
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AN 80056730 MEDLINE

L147 ANSWER 130 OF 141 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
DUPLICATE 90
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SO Proceedings of the National Academy of Sciences of the United States of America, (1979) Vol. 76, No. 4, pp. 1911-1915.
CODEN: PNASA6. ISSN: 0027-8424.
AU ROBBINS A R [Reprint author]
AN 1979:232092 BIOSIS

L147 ANSWER 131 OF 141 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED. on STN DUPLICATE 91
TI Extracellular accumulation of **lysosomal hydrolases** from astrocytes in vitro.
SO IRCS Medical Science, (1979) 7/10 (511).
CODEN: IRLCDZ
AU McCormick D.; Wallace I.
AN 79240642 EMBASE

L147 ANSWER 132 OF 141 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED. on STN DUPLICATE 92
TI Urinary **lysosomal hydrolases** in mucolipidosis II and mucolipidosis III.
SO Biochemical Journal, (1979) 177/2 (409-415).
CODEN: BIJOAK
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AN 79142350 EMBASE

L147 ANSWER 133 OF 141 MEDLINE on STN
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L147 ANSWER 134 OF 141 MEDLINE on STN
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Journal code: 7605701. ISSN: 0361-7742.
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AN 78202238 MEDLINE

L147 ANSWER 135 OF 141 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED. on STN
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AN 78255372 EMBASE

L147 ANSWER 136 OF 141 MEDLINE on STN DUPLICATE 93
TI Carbohydrate composition of purified serum glycoproteins in mucolipidosis II and mucolipidosis III.
SO Molecular and cellular biochemistry, (1978 Oct 13) 21 (1) 17-21.
Journal code: 0364456. ISSN: 0300-8177.
AU Freeze H; Kress B C; Williams J C; Cerda-Ruiz M; Miller A L
AN 79093680 MEDLINE

L147 ANSWER 137 OF 141 MEDLINE on STN DUPLICATE 94
TI Phosphohexosyl components of a lysosomal enzyme are recognized by pinocytosis receptors on human fibroblasts.
SO Proceedings of the National Academy of Sciences of the United States of America, (1977 May) 74 (5) 2026-30.
Journal code: 7505876. ISSN: 0027-8424.
AU Kaplan A; Achord D T; Sly W S
AN 77193975 MEDLINE

L147 ANSWER 138 OF 141 HCAPLUS COPYRIGHT 2004 ACS on STN
TI Interaction between lysosomal glycosidases from human plasma and lectins
SO Protides of the Biological Fluids (1976), Volume Date 1975, 23, 659-62
CODEN: PBFPA6; ISSN: 0079-7065
AU Kint, J. A.
AN 1976:518763 HCAPLUS
DN 85:118763

L147 ANSWER 139 OF 141 MEDLINE on STN DUPLICATE 95
TI Effects of metabolites present during growth of Tetrahymena pyriformis on the subsequent secretion of **lysosomal hydrolases.**
SO Journal of cellular physiology, (1975 Aug) 86 (1) 131-42.
Journal code: 0050222. ISSN: 0021-9541.
AU Blum J J
AN 76025202 MEDLINE

L147 ANSWER 140 OF 141 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN DUPLICATE 96
TI EFFECTS OF CONCANAVALIN A ON MOUSE PERITONEAL MACROPHAGES PART 2 METABOLISM OF ENDOCYTIZED PROTEINS AND REVERSIBILITY OF THE EFFECTS BY **MANNOSE.**
SO Journal of Experimental Medicine, (1974) Vol. 140, No. 5, pp. 1387-1403.
CODEN: JEMEAV. ISSN: 0022-1007.
AU EDELSON P J; COHN Z A
AN 1975:125429 BIOSIS

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L147 ANSWER 141 OF 141  HCAPLUS  COPYRIGHT 2004 ACS on STN
TI   Lysosomal hydrolases as glycoproteins
SO   Life Sciences (1970), 9(23)(Pt. 2), 1341-50
     CODEN: LIFSAK; ISSN: 0024-3205
AU   Goldstone, A.; Koenig, H.
AN   1971:72048  HCAPLUS
DN   74:72048

=> s lysosom?(5a)target?
FILE 'MEDLINE'
         39021 LYSOSOM?
        264684 TARGET?
L148       735 LYSOSOM?(5A)TARGET?

FILE 'SCISEARCH'
         22909 LYSOSOM?
        305972 TARGET?
L149       694 LYSOSOM?(5A)TARGET?

FILE 'LIFESCI'
          7194 LYSOSOM?
         99255 TARGET?
L150       275 LYSOSOM?(5A)TARGET?

FILE 'BIOTECHDS'
           488 LYSOSOM?
         23355 TARGET?
L151        45 LYSOSOM?(5A)TARGET?

FILE 'BIOSIS'
         38506 LYSOSOM?
        260738 TARGET?
L152       808 LYSOSOM?(5A)TARGET?

FILE 'EMBASE'
         30316 LYSOSOM?
        255838 TARGET?
L153       692 LYSOSOM?(5A)TARGET?

FILE 'HCAPLUS'
         33872 LYSOSOM?
        372304 TARGET?
L154       894 LYSOSOM?(5A)TARGET?

FILE 'NTIS'
           280 LYSOSOM?
         62439 TARGET?
L155        10 LYSOSOM?(5A)TARGET?

FILE 'ESBIOBASE'
          8550 LYSOSOM?
        155497 TARGET?
L156       446 LYSOSOM?(5A)TARGET?

FILE 'BIOTECHNO'
          8722 LYSOSOM?
        111737 TARGET?
L157       431 LYSOSOM?(5A)TARGET?

FILE 'WPIDS'
           600 LYSOSOM?
        137309 TARGET?
L158        34 LYSOSOM?(5A)TARGET?
```

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TOTAL FOR ALL FILES
L159       5064 LYSOSOM?(5A) TARGET?

=> s l159 and lectin#
FILE 'MEDLINE'
         37257 LECTIN#
L160        21 L148 AND LECTIN#

FILE 'SCISEARCH'
         26124 LECTIN#
L161        14 L149 AND LECTIN#

FILE 'LIFESCI'
          8887 LECTIN#
L162         2 L150 AND LECTIN#

FILE 'BIOTECHDS'
          1117 LECTIN#
L163         1 L151 AND LECTIN#

FILE 'BIOSIS'
         32677 LECTIN#
L164        10 L152 AND LECTIN#

FILE 'EMBASE'
         23005 LECTIN#
L165        11 L153 AND LECTIN#

FILE 'HCAPLUS'
         36038 LECTIN#
L166        18 L154 AND LECTIN#

FILE 'NTIS'
           140 LECTIN#
L167         0 L155 AND LECTIN#

FILE 'ESBIOBASE'
          8314 LECTIN#
L168         8 L156 AND LECTIN#

FILE 'BIOTECHNO'
          9786 LECTIN#
L169         9 L157 AND LECTIN#

FILE 'WPIDS'
          2036 LECTIN#
L170         1 L158 AND LECTIN#

TOTAL FOR ALL FILES
L171        95 L159 AND LECTIN#

=> s l159 and (mannose or m6p)
FILE 'MEDLINE'
         17284 MANNOSE
           196 M6P
L172       204 L148 AND (MANNOSE OR M6P)

FILE 'SCISEARCH'
         12985 MANNOSE
           217 M6P
L173       174 L149 AND (MANNOSE OR M6P)

FILE 'LIFESCI'
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          5734 MANNOSE
            66 M6P
L174         74 L150 AND (MANNOSE OR M6P)

FILE 'BIOTECHDS'
          1672 MANNOSE
             8 M6P
L175          8 L151 AND (MANNOSE OR M6P)

FILE 'BIOSIS'
         19914 MANNOSE
           250 M6P
L176       206 L152 AND (MANNOSE OR M6P)

FILE 'EMBASE'
         13513 MANNOSE
           184 M6P
L177       178 L153 AND (MANNOSE OR M6P)

FILE 'HCAPLUS'
         35664 MANNOSE
           235 M6P
L178       233 L154 AND (MANNOSE OR M6P)

FILE 'NTIS'
           112 MANNOSE
             8 M6P
L179          0 L155 AND (MANNOSE OR M6P)

FILE 'ESBIOBASE'
          5153 MANNOSE
           131 M6P
L180         97 L156 AND (MANNOSE OR M6P)

FILE 'BIOTECHNO'
          7168 MANNOSE
           113 M6P
L181       119 L157 AND (MANNOSE OR M6P)

FILE 'WPIDS'
          2583 MANNOSE
            15 M6P
L182          8 L158 AND (MANNOSE OR M6P)

TOTAL FOR ALL FILES
L183      1301 L159 AND (MANNOSE OR M6P)

=> s l183 and (recombinant# or gene/q)
FILE 'MEDLINE'
        226842 RECOMBINANT#
L184        99 L172 AND (RECOMBINANT# OR GENE/Q)

FILE 'SCISEARCH'
        135263 RECOMBINANT#
L185        75 L173 AND (RECOMBINANT# OR GENE/Q)

FILE 'LIFESCI'
         62590 RECOMBINANT#
L186        22 L174 AND (RECOMBINANT# OR GENE/Q)

FILE 'BIOTECHDS'
         81914 RECOMBINANT#
L187         7 L175 AND (RECOMBINANT# OR GENE/Q)
```

```
FILE 'BIOSIS'
        177655 RECOMBINANT#
L188         55 L176 AND (RECOMBINANT# OR GENE/Q)

FILE 'EMBASE'
        148705 RECOMBINANT#
L189         72 L177 AND (RECOMBINANT# OR GENE/Q)

FILE 'HCAPLUS'
        160669 RECOMBINANT#
L190         72 L178 AND (RECOMBINANT# OR GENE/Q)

FILE 'NTIS'
          1547 RECOMBINANT#
L191          0 L179 AND (RECOMBINANT# OR GENE/Q)

FILE 'ESBIOBASE'
         71866 RECOMBINANT#
L192         37 L180 AND (RECOMBINANT# OR GENE/Q)

FILE 'BIOTECHNO'
        126976 RECOMBINANT#
L193         58 L181 AND (RECOMBINANT# OR GENE/Q)

FILE 'WPIDS'
         34804 RECOMBINANT#
L194          8 L182 AND (RECOMBINANT# OR GENE/Q)

TOTAL FOR ALL FILES
L195        505 L183 AND (RECOMBINANT# OR GENE/Q)

=> s (l171 or l195) not 2002-2004/py
FILE 'MEDLINE'
       1314032 2002-2004/PY
L196         96 (L160 OR L184) NOT 2002-2004/PY

FILE 'SCISEARCH'
       2399042 2002-2004/PY
L197         66 (L161 OR L185) NOT 2002-2004/PY

FILE 'LIFESCI'
        214244 2002-2004/PY
L198         20 (L162 OR L186) NOT 2002-2004/PY

FILE 'BIOTECHDS'
         53433 2002-2004/PY
L199          1 (L163 OR L187) NOT 2002-2004/PY

FILE 'BIOSIS'
       1231965 2002-2004/PY
L200         49 (L164 OR L188) NOT 2002-2004/PY

FILE 'EMBASE'
       1099415 2002-2004/PY
L201         66 (L165 OR L189) NOT 2002-2004/PY

FILE 'HCAPLUS'
       2499762 2002-2004/PY
L202         58 (L166 OR L190) NOT 2002-2004/PY

FILE 'NTIS'
         27941 2002-2004/PY
L203          0 (L167 OR L191) NOT 2002-2004/PY
```

FILE 'ESBIOBASE'
683260 2002-2004/PY
L204 31 (L168 OR L192) NOT 2002-2004/PY

FILE 'BIOTECHNO'
244553 2002-2004/PY
L205 56 (L169 OR L193) NOT 2002-2004/PY

FILE 'WPIDS'
2484316 2002-2004/PY
L206 0 (L170 OR L194) NOT 2002-2004/PY

TOTAL FOR ALL FILES
L207 443 (L171 OR L195) NOT 2002-2004/PY

=> dup rem l207
PROCESSING COMPLETED FOR L207
L208 143 DUP REM L207 (300 DUPLICATES REMOVED)

=> d tot

L208 ANSWER 1 OF 143 SCISEARCH COPYRIGHT 2004 THOMSON ISI on STN
TI Biodistribution, kinetics, and efficacy of highly phosphorylated and non-phosphorylated beta-glucuronidase in the murine model of mucopolysaccharidosis VII
SO JOURNAL OF BIOLOGICAL CHEMISTRY, (16 NOV 2001) Vol. 276, No. 46, pp. 43160-43165.
Publisher: AMER SOC BIOCHEMISTRY MOLECULAR BIOLOGY INC, 9650 ROCKVILLE PIKE, BETHESDA, MD 20814 USA.
ISSN: 0021-9258.
AU Sands M S (Reprint); Vogler C A; Ohlemiller K K; Roberts M S; Grubb J H; Levy B; Sly W S
AN 2001:919950 SCISEARCH

L208 ANSWER 2 OF 143 MEDLINE on STN DUPLICATE 1
TI Human acid ceramidase: processing, glycosylation, and **lysosomal targeting.**
SO Journal of biological chemistry, (2001 Sep 21) 276 (38) 35352-60.
Journal code: 2985121R. ISSN: 0021-9258.
AU Ferlinz K; Kopal G; Bernardo K; Linke T; Bar J; Breiden B; Neumann U; Lang F; Schuchman E H; Sandhoff K
AN 2001527195 MEDLINE

L208 ANSWER 3 OF 143 MEDLINE on STN
TI Binding of GGA2 to the lysosomal enzyme sorting motif of the **mannose** 6-phosphate receptor.
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Journal code: 0404511. ISSN: 0036-8075.
AU Zhu Y; Doray B; Poussu A; Lehto V P; Kornfeld S
AN 2001314374 MEDLINE

L208 ANSWER 4 OF 143 MEDLINE on STN DUPLICATE 2
TI Enzyme therapy for lysosomal acid lipase deficiency in the mouse.
SO Human molecular genetics, (2001 Aug 1) 10 (16) 1639-48.
Journal code: 9208958. ISSN: 0964-6906.
AU Du H; Schiavi S; Levine M; Mishra J; Heur M; Grabowski G A
AN 2001441564 MEDLINE

L208 ANSWER 5 OF 143 MEDLINE on STN DUPLICATE 3
TI Adenovirus serotype 7 retention in a late endosomal compartment prior to cytosol escape is modulated by fiber protein.
SO Journal of virology, (2001 Feb) 75 (3) 1387-400.
Journal code: 0113724. ISSN: 0022-538X.
AU Miyazawa N; Crystal R G; Leopold P L

AN 2001111656 MEDLINE

L208 ANSWER 6 OF 143 MEDLINE on STN DUPLICATE 4
TI Overexpression of a rat kinase-deficient phosphoinositide 3-kinase, Vps34p, inhibits cathepsin D maturation.
SO Biochemical journal, (2001 Feb 1) 353 (Pt 3) 655-61.
Journal code: 2984726R. ISSN: 0264-6021.
AU Row P E; Reaves B J; Domin J; Luzio J P; Davidson H W
AN 2001194977 MEDLINE

L208 ANSWER 7 OF 143 MEDLINE on STN
TI Mutations of the C-terminal end of cathepsin K affect proenzyme secretion and intracellular maturation.
SO Biochemical and biophysical research communications, (2001 Feb 23) 281 (2) 551-7.
Journal code: 0372516. ISSN: 0006-291X.
AU Claveau D; Riendeau D
AN 2001182379 MEDLINE

L208 ANSWER 8 OF 143 MEDLINE on STN DUPLICATE 5
TI **Mannose** 6-phosphate-independent endocytosis of beta-glucuronidase by human fibroblasts. I. Evidence for the existence of a membrane-binding activity.
SO Biochimica et biophysica acta, (2001 Apr 23) 1538 (2-3) 141-51.
Journal code: 0217513. ISSN: 0006-3002.
AU Gonzalez-Noriega A; Michalak C; Cruz-Perez J R; Masso F
AN 2001341378 MEDLINE

L208 ANSWER 9 OF 143 SCISEARCH COPYRIGHT 2004 THOMSON ISI on STN
TI **Mannose** 6-phosphate-independent endocytosis of beta-glucuronidase by human fibroblasts I. evidence for the existence of a membrane-binding activity
SO BIOCHIMICA ET BIOPHYSICA ACTA-MOLECULAR CELL RESEARCH, (23 APR 2001) Vol. 1538, No. 2-3, pp. 141-151.
Publisher: ELSEVIER SCIENCE BV, PO BOX 211, 1000 AE AMSTERDAM, NETHERLANDS.
ISSN: 0167-4889.
AU Gonzalez-Noriega A (Reprint); Michalak C; Cruz-Perez J R; Masso F
AN 2001:387562 SCISEARCH

L208 ANSWER 10 OF 143 SCISEARCH COPYRIGHT 2004 THOMSON ISI on STN
TI Distinct reading of different structural determinants modulates the dileucine-mediated transport steps of the lysosomal membrane protein LIMPII and the insulin-sensitive glucose transporter GLUT4
SO JOURNAL OF BIOLOGICAL CHEMISTRY, (22 DEC 2000) Vol. 275, No. 51, pp. 39874-39885.
Publisher: AMER SOC BIOCHEMISTRY MOLECULAR BIOLOGY INC, 9650 ROCKVILLE PIKE, BETHESDA, MD 20814 USA.
ISSN: 0021-9258.
AU Sandoval I V (Reprint); Martinez-Arca S; Valdueza J; Palacios S; Holman G D
AN 2001:39472 SCISEARCH

L208 ANSWER 11 OF 143 MEDLINE on STN DUPLICATE 6
TI Identification of a novel **sequence** involved in lysosomal sorting of the sphingolipid activator protein prosaposin.
SO Journal of biological chemistry, (2000 Aug 11) 275 (32) 24829-39.
Journal code: 2985121R. ISSN: 0021-9258.
AU Zhao Q; Morales C R
AN 2000428439 MEDLINE

L208 ANSWER 12 OF 143 LIFESCI COPYRIGHT 2004 CSA on STN DUPLICATE 7
TI Live Salmonella Modulate Expression of Rab Proteins to Persist in a Specialized Compartment and Escape Transport to Lysosomes

SO Journal of Biological Chemistry [J. Biol. Chem.], (20000526) vol. 275, no. 21, pp. 16281-16288.
ISSN: 0021-9258.
AU Hashim, S.; Mukherjee, K.; Raje, M.; Basu, S.K.; Mukhopadhyay, A.
AN 2001:28529 LIFESCI

L208 ANSWER 13 OF 143 MEDLINE on STN DUPLICATE 8
TI Targeted disruption of the cation-dependent or cation-independent **mannose** 6-phosphate receptor does not decrease the content of acid glycosidases in the acrosome.
SO Journal of andrology, (2000 Nov-Dec) 21 (6) 944-53.
Journal code: 8106453. ISSN: 0196-3635.
AU Chayko C A; Orgebin-Crist M C
AN 2001303904 MEDLINE

L208 ANSWER 14 OF 143 MEDLINE on STN DUPLICATE 9
TI The dendritic cell receptor for endocytosis, DEC-205, can recycle and enhance antigen presentation via major histocompatibility complex class II-positive lysosomal compartments.
SO Journal of cell biology, (2000 Oct 30) 151 (3) 673-84.
Journal code: 0375356. ISSN: 0021-9525.
AU Mahnke K; Guo M; Lee S; Sepulveda H; Swain S L; Nussenzweig M; Steinman R M
AN 2001042083 MEDLINE

L208 ANSWER 15 OF 143 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
TI Lysosomal fusion following FcgammaRIIA phagocytosis is mediated by a novel cytoplasmic motif.
SO Blood, (November 16, 2000) Vol. 96, No. 11 Part 1, pp. 443a. print.
Meeting Info.: 42nd Annual Meeting of the American Society of Hematology. San Francisco, California, USA. December 01-05, 2000. American Society of Hematology.
CODEN: BLOOAW. ISSN: 0006-4971.
AU Worth, Randall G. [Reprint author]; Kim, Moo-kyung [Reprint author]; Mayo-Bond, Laura; Todd, Robert F., III; Petty, Howard R.; Schreiber, Alan D. [Reprint author]
AN 2001:314511 BIOSIS

L208 ANSWER 16 OF 143 MEDLINE on STN DUPLICATE 10
TI "Supercharged Cells" for delivery of **recombinant** human iduronate-2-sulfatase.
SO Molecular genetics and metabolism, (2000 Jul) 70 (3) 170-8.
Journal code: 9805456. ISSN: 1096-7192.
AU Pan D; Jonsson J J; Braun S E; McIvor R S; Whitley C B
AN 2000418896 MEDLINE

L208 ANSWER 17 OF 143 MEDLINE on STN DUPLICATE 11
TI Protease trafficking in two primitive eukaryotes is mediated by a prodomain protein motif.
SO Journal of biological chemistry, (1999 Jun 4) 274 (23) 16249-56.
Journal code: 2985121R. ISSN: 0021-9258.
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AN 1999278392 MEDLINE

L208 ANSWER 18 OF 143 MEDLINE on STN DUPLICATE 12
TI Altered ligand binding by insulin-like growth factor II/**mannose** 6-phosphate receptors bearing missense mutations in human cancers.
SO Cancer research, (1999 Sep 1) 59 (17) 4314-9.
Journal code: 2984705R. ISSN: 0008-5472.
AU Devi G R; De Souza A T; Byrd J C; Jirtle R L; MacDonald R G
AN 1999413488 MEDLINE

L208 ANSWER 19 OF 143 MEDLINE on STN
TI Interaction of arylsulfatase A with UDP-N-acetylglucosamine:Lysosomal

enzyme-N-acetylglucosamine-1-phosphotransferase.
SO Journal of biological chemistry, (1999 Feb 5) 274 (6) 3651-8.
Journal code: 2985121R. ISSN: 0021-9258.
AU Schierau A; Dietz F; Lange H; Schestag F; Parastar A; Gieselmann V
AN 1999121106 MEDLINE

L208 ANSWER 20 OF 143 MEDLINE on STN DUPLICATE 13
TI Human dendritic cells shed a functional, soluble form of the mannose receptor.
SO International immunology, (1999 Nov) 11 (11) 1775-80.
Journal code: 8916182. ISSN: 0953-8178.
AU Jordens R; Thompson A; Amons R; Koning F
AN 2000014651 MEDLINE

L208 ANSWER 21 OF 143 MEDLINE on STN DUPLICATE 14
TI Alternative mechanisms for trafficking of lysosomal enzymes in **mannose** 6-phosphate receptor-deficient mice are cell type-specific.
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Journal code: 0052457. ISSN: 0021-9533.
AU Dittmer F; Ulbrich E J; Hafner A; Schmahl W; Meister T; Pohlmann R; von Figura K
AN 1999230346 MEDLINE

L208 ANSWER 22 OF 143 MEDLINE on STN DUPLICATE 15
TI **Targeted** disruption of the **lysosomal** alpha-mannosidase **gene** results in mice resembling a mild form of human alpha-mannosidosis.
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Journal code: 9208958. ISSN: 0964-6906.
AU Stinchi S; Lullmann-Rauch R; Hartmann D; Coenen R; Beccari T; Orlacchio A; von Figura K; Saftig P
AN 1999330543 MEDLINE

L208 ANSWER 23 OF 143 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
TI The IGF 2 receptor and **targeting** of **lysosomal** enzymes: Implications for intracellular degradation of IGFBP-3.
SO Growth Hormone and IGF Research, (Oct., 1999) Vol. 9, No. 5, pp. 330-331. print.
Meeting Info.: 5th International Symposium on Insulin-Like Growth Factors. Brighton, England, UK. October 31-November 4, 1999. Growth Hormone Research Society.
ISSN: 1096-6374.
AU Braulke, T. [Reprint author]; Storch, S. [Reprint author]; Dittmer, F.; Goetz, W.
AN 2000:28099 BIOSIS

L208 ANSWER 24 OF 143 MEDLINE on STN DUPLICATE 16
TI The mammalian AP-3 adaptor-like complex mediates the intracellular transport of lysosomal membrane glycoproteins.
SO Journal of biological chemistry, (1998 Nov 6) 273 (45) 29451-61.
Journal code: 2985121R. ISSN: 0021-9258.
AU Le Borgne R; Alconada A; Bauer U; Hoflack B
AN 1999009051 MEDLINE

L208 ANSWER 25 OF 143 MEDLINE on STN DUPLICATE 17
TI Purification and multimeric structure of bovine N-acetylglucosamine-1-phosphodiester alpha-N-acetylglucosaminidase.
SO Journal of biological chemistry, (1998 Sep 4) 273 (36) 23203-10.
Journal code: 2985121R. ISSN: 0021-9258.
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L208 ANSWER 139 OF 143 MEDLINE on STN
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L208 ANSWER 140 OF 143 MEDLINE on STN DUPLICATE 85
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L208 ANSWER 143 OF 143 MEDLINE on STN DUPLICATE 86
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L209   23 L148 AND GLUCOSIDASE#

FILE 'SCISEARCH'
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L210   22 L149 AND GLUCOSIDASE#

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L218   12 L157 AND GLUCOSIDASE#

FILE 'WPIDS'
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L222         16 L210 NOT 2002-2004/PY

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L223          6 L211 NOT 2002-2004/PY

FILE 'BIOTECHDS'
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L224          1 L212 NOT 2002-2004/PY

FILE 'BIOSIS'
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L225         21 L213 NOT 2002-2004/PY

FILE 'EMBASE'
       1099415 2002-2004/PY
L226         14 L214 NOT 2002-2004/PY

FILE 'HCAPLUS'
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L227         18 L215 NOT 2002-2004/PY

FILE 'NTIS'
         27941 2002-2004/PY
L228          0 L216 NOT 2002-2004/PY

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L229          9 L217 NOT 2002-2004/PY

FILE 'BIOTECHNO'
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L230         10 L218 NOT 2002-2004/PY

FILE 'WPIDS'
       2484316 2002-2004/PY
L231          0 L219 NOT 2002-2004/PY

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L232        112 L220 NOT 2002-2004/PY

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